2.7

Lower Duwamish Waterway Group

Port of Seattle | City of Seattle | King County | The Boeing Company

Lower Duwamish Waterway Remedial Investigation

APPENDIX A. PHASE 1 ECOLOGICAL RISK ASSESSMENT

FINAL

For submittal to

The U.S. Environmental Protection Agency
Region 10
Seattle, WA

The Washington State Department of Ecology
Northwest Regional Office
Bellevue, WA

July 3, 2003

Prepared by: Windward environmental LLC

200 West Mercer Street, Suite 401 • Seattle, Washington • 98119

Table of Contents

List of Tables[1]

[1] Includes figures from King County WQA (Attachment A.2)

List of Figures

GIS map titles listed here for reference; maps published as a separate document

Acronyms

Acronym	definition
AChE	acetylcholine esterase
ACOE	US Army Corps of Engineers
AET	apparent effects threshold
AHH	aryl hydrocarbon hydroxylase
ATSDR	Agency for Toxic Substance and Disease Registry
AWQC	ambient water quality criteria
BAF	bioaccumulation factor
BaP	benzo(a)pyrene
BCF	bioconcentration factor
BEHP	bis(2-ethylhexyl)phthalate
BSAF	biota-sediment accumulation factor
bw	body weight
COPC	chemical of potential concern
CSL	cleanup screening level of SMS
CSO	combined sewer overflow
DDTs	DDT and its metabolites
DMMP	Dredged Material Management Program
dw	dry weight
EEC	estimated exposure concentration
EED	estimated environmental dose
EPA	U.S. Environmental Protection Agency
ERA	ecological risk assessment
ESA	Endangered Species Act
FAC	fluorescent aromatic compound
FMR	free-living metabolic rate
GIS	geographic information system
HPAH	high-molecular-weight polycyclic aromatic hydrocarbon
HQ	hazard quotient
IP	intraperitoneal
LDW	Lower Duwamish Waterway
LDWSI	LDW Site Inspection
LOAEL	lowest-observed-apparent-effects level
LOEC	lowest-observed-effects concentration
LPAH	low-molecular-weight polycyclic aromatic hydrocarbon
MHHW	mean higher high water
ML	maximum level in DMMP
MLLW	mean lower low water
NOAEL	no-observed-apparent-effects level
NOEC	no-observed-effects concentration
OC	organic carbon

Acronym	definition
PAH	polycyclic aromatic hydrocarbon
PCB	polychlorinated biphenyl
PCDD	polychlorinated dibenzodioxin
PCDF	polychlorinated dibenzofuran
PPF	predator-prey factor
PSDDA	Puget Sound Dredged Disposal Analysis
QSAR	quantitative structure-activity relationship
RI	Remedial Investigation
RI/FS	Remedial Investigation/Feasibility Study
RK	river kilometer
RM	river mile
ROC	receptor of concern
SL	screening level in DMMP
SMS	Washington State Sediment Management Standards
SOW	statement of work
SQS	sediment quality standards of SMS
SUF	site usage factor
SVOC	semivolatile organic compound
SWA	spatially weighted average
T&E	threatened or endangered
TBT	tributyltin
TBTO	tributyltin oxide
TCDD	tetrachlorodibenzo-p-dioxin
TOC	total organic carbon
TRV	toxicity reference value
UCL	upper confidence limit
VOC	volatile organic compound
WDFW	Washington Department of Fish and Wildlife
WQA	(King County) Combined Sewer Overflow Water Quality Assessment
ww	wet weight

Executive Summary

This appendix contains the Phase 1 ecological risk assessment (ERA) for the Lower Duwamish Waterway (LDW). Using existing data, the Phase 1 ERA evaluated risks from sediment-associated chemicals to benthic invertebrates, fish, and wildlife species that may use the LDW for habitat and food for at least a portion of their life span. Although there is relatively little suitable habitat presently available for rooted aquatic plants within the LDW, risks to this group were also evaluated. Ecological risks in the LDW are being assessed through a two-phase process. The Phase 1 ERA, presented in this appendix, provides:

- Preliminary risk estimates based on available data for ecological receptors of concern (ROCs) from chemicals of potential concern (COPCs)
- A forum for communication and input from stakeholders regarding key risk issues and approaches
- A list of uncertainties, including their potential impact on risk conclusions, to form the basis for the identification of data gaps[2] that may need to be filled prior to completion of the Phase 2 ERA
- Risk-based analyses to aid in the identification of high priority sites for the candidate early action site process (Windward 2002)
- As part of the Phase 2 (baseline) ERA, which will be initiated in 2003, additional field data will be collected to fill critical data gaps identified in Phase 1. These data will be combined with existing field and analytical data to reevaluate risk conclusions made in this Phase 1 ERA,[3] to assess risks to ecological receptors in the absence of any early actions, and to estimate risks at the site following completion of early remedial actions (i.e., residual risk). The Phase 2 ERA will be used to support remedial decision-making at the site, and will be contained in its entirety in the Phase 2 RI.
- This executive summary contains a brief summary of each of the major components of the ERA including the problem formulation, the exposure assessment, the effects assessment, and the risk characterization and uncertainty assessment.

---

[2] In the data gaps memorandum (Final Draft to to be submitted in 2003) uncertainties identified in the Phase 1 ERA, human health risk assessment (HHRA), and RI are being evaluated to determine what additional analyses (primarily fieldwork) should be conducted prior to the Phase 2 ERA.

[3] Phase 1 risk conclusions include the COPC screen in the problem formulation and the hazard quotients (HQs) calculated in the Phase 1 risk characterization. The reevaluation of these risk conclusions in the Phase 2 ERA is necessary because of the limited tissue dataset available in the Phase 1 ERA, and thus the preliminary nature of many of the results.

ES.1 PROBLEM FORMULATION

The problem formulation of the ERA establishes the overall scope of the assessment. Because it is impractical to evaluate every potentially-exposed species, it is standard ERA practice to focus on representative receptor species that typify groups of organisms with specific exposure pathways. One objective of selecting representative receptors is to choose species for which the risk conclusions will be protective of other species that are not explicitly evaluated. For example, an assessment of great blue heron risk would be assumed to be protective of all wading birds that eat fish. Decisions on species inclusion and assumptions on exposure parameters are deliberately biased in an environmentally conservative manner to ensure a protective assessment. In addition, risks to some species are analyzed because those species are highly valued by society, such as endangered or threatened species.

Representative ROCs selected for this Phase 1 ERA were benthic invertebrates, crabs, English sole, great blue heron, spotted sandpiper, bald eagle, river otter, harbor seal, and aquatic rooted plants. In addition, juvenile chinook salmon and bull trout were selected as ROCs because they are federally protected species with complete exposure pathways in the LDW.

For each representative species selected, COPCs were identified. An initial screening conducted in the problem formulation, using highly conservative assumptions, identified 59 chemicals (including tributyltin [TBT], metals, polychlorinated biphenyls [PCBs] and other organic compounds) as COPCs for benthic invertebrates and crabs, 7 chemicals (PCBs, polycyclic aromatic hydrocarbons [PAHs], TBT, DDT, arsenic, copper, and mercury) as COPCs for at least one fish species, and 7 chemicals (PCBs, bis[2-ethylhexyl]phthalate, arsenic, copper, lead, mercury, and zinc) as COPCs for at least one wildlife species.

In addition, conceptual site models were developed to identify complete exposure pathways for COPCs from sources to representative species, and assessment and measurement endpoints were identified in the problem formulation. The representative species, COPCs, pathways, and endpoints formed the scope for the remainder of the Phase 1 ERA. Uncertainties associated with these analyses were acknowledged, and reserved for further discussion in the uncertainty assessment.

ES.2 EXPOSURE ASSESSMENT

To refine the initial risk-based screening conducted in the problem formulation, more detailed analyses were conducted in the exposure assessment to conservatively estimate the potential exposure of each ROC to the sediment-associated COPCs identified in the problem formulation. Exposure of benthic invertebrates to COPCs was primarily assessed by evaluating the distribution, concentration, and co-occurrence of COPCs in surface sediment, with the exception of risk to crab and risk from sediment-associated TBT, which were both assessed using a tissue residue approach. Exposure of fish or wildlife to COPCs was either characterized from tissue

body burden data or from estimated dietary exposure doses. Dietary ingestion was estimated through consideration of available information on ROC life histories, including body weight, feeding behavior, diet, and relationship to the aquatic food web. Surface sediment data in intertidal and marsh areas were used to estimate exposure of aquatic rooted plants to COPCs.

ES.3 Effects Assessment

Potential adverse effects of exposure (i.e., mortality, reduced growth, or impaired reproduction) were assessed in the effects assessment. Sediment quality standards and guidelines were used to predict potential effects in benthic invertebrates. For crabs, fish, wildlife, and plants, a detailed evaluation of studies in the scientific literature documenting effects of COPCs on the ROCs or similar species was conducted. This literature review identified COPC concentrations (or doses where appropriate) associated with no effects (i.e., safe concentrations or doses), in addition to concentrations (or doses) documented to cause adverse effects. Both sets (i.e., lowest observed effect concentration [LOEC] and no observed effect concentration [NOEC]) of toxicity reference values (TRVs) were summarized in tables and the rationale for TRV selection provided. Available site-specific effects data (e.g., sediment toxicity tests) were also discussed, although chemical-specific TRVs could not be derived from these data, due in part to their limited availability.

ES.4 Risk Characterization and Uncertainty Assessment

The exposure and effects data were compared in the risk characterization to assess the potential for sediment-associated COPCs to cause adverse effects to ROCs. All assessments of exposure and risk were intentionally conservatively biased to minimize the possibility of a false negative finding (i.e., predicting an absence of toxicity when, in fact, there would be a toxic effect). However, the exposure assessments used more realistic assumptions (e.g., exposure estimated using spatially weighted average concentrations vs. maximum concentrations for receptors with large home ranges) than were used in the problem formulation. Based on available data, this analysis identified the following Phase 1 conclusions:

- **Benthic invertebrates**—Sixty COPCs (including TBT) were identified for benthic invertebrates based on a comparison of sediment data to sediment quality guidelines and standards. Risk to crab from COPCs, with the possible exception of arsenic, appears to be low based on existing data.
- **Fish**—Exposure concentrations for three of the seven COPCs (arsenic, copper, and PCBs) were greater than concentrations associated with adverse effects for one or more fish ROCs. Three COPCs (PAHs, mercury, and TBT) had exposure estimates exceeding a no-effects level, but lower than adverse effect levels associated with survival, growth, or reproduction. None of the DDT exposure estimates exceeded either effects or no-effects levels for any of the fish ROCs.

- **Wildlife**—None of the COPCs had dietary exposure estimates greater than doses associated with adverse effects to survival, growth or reproduction for any of the wildlife ROCs. In contrast, preliminary risk estimates of PCBs to great blue herons using egg data indicated that exposure may be occurring at levels associated with adverse effects. Four of the seven COPCs (lead, mercury, arsenic, and PCBs) had exposure estimates greater than no-effects levels for one or more of the wildlife ROCs.
- **Rooted Aquatic Plants**—Of the four COPCs evaluated for plants (lead, mercury, PCBs, and zinc), marsh sediment concentrations were less than soil PCB concentrations associated with no effects, but were within the low end of the concentration range associated with effects for lead and zinc.[4] Due to the uncertainty associated with the effects data, risk estimates for plants are highly uncertain, but in general are much lower that that predicted based on background concentrations in marsh areas.

Regional and natural background issues regarding arsenic for fish and wildlife will be discussed in the Phase 2 risk characterization, per EPA (2002) guidance. Based on results of the Phase 1 RI and RAs and discussions with the agencies and stakeholders, a data gaps memorandum (final draft to be submitted in 2003) is being produced as part of the overall Phase 1 RI process. This memorandum assesses the feasibility of gathering additional site-specific data, and how valuable that information would be in reducing uncertainty in risk estimates. Additional data collected to fill gaps identified in the data gaps memorandum will be fully evaluated in the Phase 2 ERA. In the Phase 2 ERA, risks associated with exposure of ecological receptors to COPCs[5] within the LDW will be quantitatively characterized in a manner designed to support sound risk management decisions.

[4] Effects data were not available for mercury.

[5] Phase 2 COPCs will be determined as part of the Phase 2 ERA problem formulation.

A.1 Introduction

This document presents the Phase 1 scoping-phase ecological risk assessment (ERA)[6] for the Lower Duwamish Waterway (LDW) in Seattle, Washington. It has been developed in accordance with both national and regional US Environmental Protection Agency (EPA) guidance (EPA 1992; 1997a,b; 1998a).

ERA is an integral part of the Remedial Investigation/Feasibility Study (RI/FS) process to support management decisions. An ERA evaluates the likelihood that adverse biological effects are occurring or may occur as a result of exposure to one or more stressors (EPA 1992).

In the LDW, risks to ecological receptors from contaminated sediments are being addressed in a tiered process consisting of the following assessments. First, the Phase 1 ERA (this document) was conducted using existing data to provide:

- Preliminary risk estimates based on available data for ecological receptors of concern (ROCs) from chemicals of potential concern (COPC)
- A forum for communication and input from stakeholders regarding key issues and approaches
- A list of uncertainties including their potential impact on risk conclusions to form the basis for the identification of data gaps[7] that may need to be filled prior to completion of the Phase 2 ERA
- Risk-based analyses to aid in the identification of high priority sites for the candidate early action site process (Windward 2003).
- Second, a Phase 2 ERA will be conducted. As part of the Phase 2 (baseline) ERA, which will be initiated in 2003, additional data will be collected to fill critical data gaps identified in Phase 1. These data will be combined with existing field and analytical data to reevaluate risk conclusions made in this Phase 1 ERA,[8] to assess risks to ecological receptors in the absence of any early actions, and to estimate risks at the site following completion of early remedial actions (i.e., residual risk). The Phase 2 ERA will be used to support remedial decision-making at the site, and will be contained in its entirety in the Phase 2 RI.

---

[6] Hereafter referred to as the Phase 1 ERA.

[7] In the data gaps memorandum, to be submitted in in final form in 2003, uncertainties identified in the Phase 1 RA, human health risk assessment (HHRA), and RI are being evaluated to determine what additional analyses (primarily field work) should be conducted prior to the Phase 2 ERA..

[8] Phase 1 risk conclusions include the COPC screen in the problem formulation (Section 2) and the hazard quotients (HQs) calculated in the Phase 1 risk characterization (Section 7). The reevaluation of these risk conclusions is necessary because of the limited tissue dataset available in the Phase 1 ERA, and thus the preliminary nature of many of the results.

- This appendix contains the Phase 1 ERA and is arranged in the following sections:
- A.2 - Problem formulation
- A.3 - Exposure and effects assessment of benthic invertebrates
- A.4 - Exposure and effects assessment of fish
- A.5 - Exposure and effects assessment of wildlife
- A.6 - Exposure and effects assessment of plants
- A.7 - Risk characterization and Uncertainty Assessment
- A.8 - Conclusions
- A.9 - References

This appendix also includes the following attachments:

- Attachment A.1 - 11×17 GIS map folio referenced in this document
- Attachment A.2 - Summary of King County Water Quality Assessment of Risks to Fish and Invertebrates in the Water Column
- Attachment A.3 - Tables and figures from the King County Combined Sewer Overflow Water Quality Assessment (WQA) Wildlife Risk Assessment (King County 1999c)

A.2 Problem Formulation

This section presents the problem formulation for the Phase 1 ERA. Through the use of a conservative screening approach, the problem formulation establishes which ROC/COCP pairs are further evaluated in the exposure and effects assessment, the risk characterization, and the uncertainty assessment in the Phase 1 ERA. This section includes information regarding the environmental setting, ecological resources that use the site, selection of ROCs, a summary of relevant available data collected from the LDW, a COPC screen for ROCs, and the conceptual site model for the LDW. Together, these elements establish the scope for this Phase 1 ERA.

A.2.1 Environmental Setting

This section presents general information about the LDW environment, history, and habitat. It provides a context for evaluating site usage (exposure) by ecological receptors and provides a background for considering non-chemical stressors. Although non-chemical stressors, such as habitat loss, can impact ecological species, this ERA is focused on sediment-associated chemical stressors in order to evaluate the likelihood of adverse ecological effects from past or ongoing releases of chemicals.

A.2.1.1 Site description and history

The Duwamish River originates at the confluence of the Black and Green Rivers near Tukwila, and subsequently flows northwesterly approximately 19 km (12 mi) into Elliott Bay in the southern Seattle waterfront. Prior to the 19th century, the Duwamish River meandered widely through a valley consisting of floodplains, freshwater wetland, and tidal marshes before emptying into Elliott Bay. Flooding was a common natural occurrence in the river valley. The Duwamish River was fed by the Green, Black, and White rivers, with a combined drainage area of approximately 4,250 km^2 (1640 mi^2) (Blomberg et al. 1988).

Today, the Green River is the main source of water into the Duwamish. The White River was diverted to the Puyallup River in 1906 to control flooding (Patmont 1983). In 1916, the Black River, which drained from Lake Washington and was fed by the Cedar River, was reduced to a minor stream when the level of Lake Washington was lowered by the construction of the Ship Canal, and the Cedar River was diverted to Lake Washington (Patmont 1983). These changes reduced the Duwamish drainage area to 1,250 km^2 (Warner and Fritz 1995). Over the past century, development and flow diversion have reduced the original drainage area of the Duwamish River by about 70%.

The LDW has been straightened and dredged in many areas by the US Army Corps of Engineers (ACOE) to facilitate navigation and industrial development. Dredging in 1903-1905 created the East and West Waterways, and dredged material from the river was used to create Harbor Island (Weston 1993). From just upstream of Turning Basin 3 to Harbor Island, the river has been dredged and channelized, and is referred to as the LDW (Figure A-2-1). From end to end, the LDW is about 8 km (5 mi) in length.

The highly developed shoreline is primarily composed of piers, riprap, constructed seawalls, and bulkheads for industrial and commercial use. The depth of the river varies from approximately 17 m (56 ft) at mean lower low water (MLLW) near the mouth to 3 m (10 ft) at MLLW (Weston 1993) near Turning Basin 3. The average width of the LDW is 134 m. The remnants of natural meanders west of Kellogg Island and along the waterway (now used as slips) are the only evidence of the river's original winding course. The former river channel and surrounding floodplains were filled and graded to form the present-day topography.

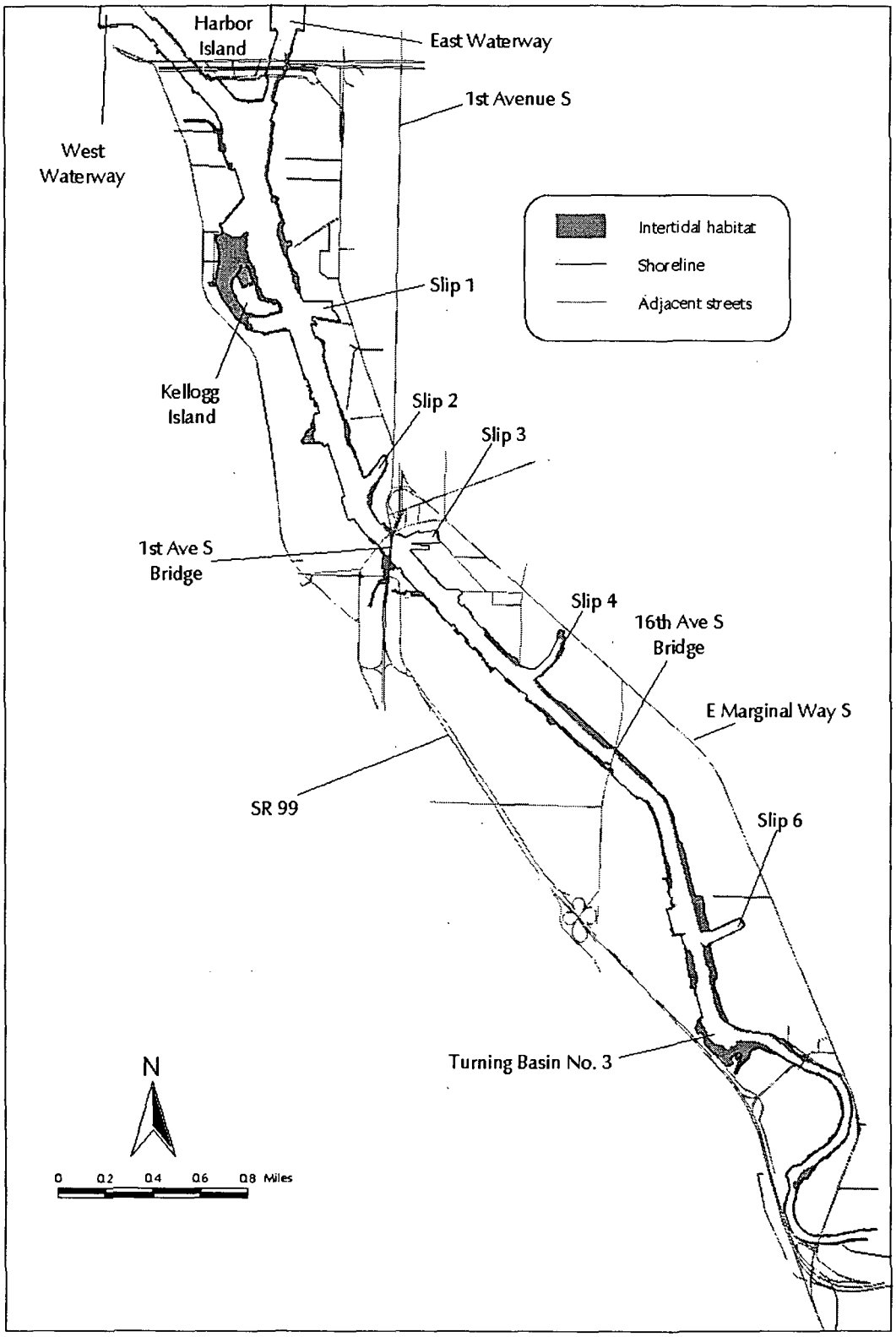

Figure A-2-1. The Lower Duwamish Waterway (LDW)

A.2.1.2 Habitat features

Sections of natural shoreline occur in the LDW only above Turning Basin 3 (Tanner 1991). Most (98%) of approximately 510 hectares (ha) (1,270 acres [ac]) of tidal marsh and 590 ha (1,450 ac) of flats and shallows, and all of about 500 ha (1,230 ac) of tidal wetland, have been either filled or dredged (Blomberg et al. 1988), or altered by the hydrologic changes discussed in A.2.1.3. Remnant tidal marshes account for only 2 ha (5 ac), and mudflats for 22 ha (54 ac) (Leon 1980). Kellogg Island, located south of Harbor Island, is surrounded by the largest remnant of intertidal habitat remaining in the LDW and is presently designated as a wildlife refuge. However, Kellogg Island has been highly altered from its historic shape and function. It was filled with dredge spoils by ACOE in the 1950s and 1960s. Present habitat associated with the island includes high and low marsh, intertidal flats, and filled uplands (Canning et al. 1979). In 1974, when the Port of Seattle deposited 1,700 m^3 (2,200 yd^3) of dredge materials on the island (Sato 1997), an upland component of Kellogg Island was created. A mixture of introduced and native plant and tree species rapidly colonized the 7-ha island.

Remnants of natural intertidal habitat occur on the northern portion of Kellogg Island and in occasional patches throughout the LDW (Figure A-2-1). The majority of the LDW shoreline is composed of riprap, pier aprons, or sheet piling (Tanner 1991). Shoreline armoring is usually present at the top of the intertidal zone, but areas of sloping mud and sandflats can exist below (Battelle et al. 2001). However, due to the shoreline armoring, these intertidal flats are partially isolated from inputs of sediment, nutrients, and organic matter (i.e., woody debris) from upland riparian vegetation zones; this isolation degrades the habitat quality of these flats (Battelle et al. 2001). In addition, overwater structures, which are common throughout the LDW, shade shallow and intertidal habitats, alter microclimates, and inhibit growth of plant communities, thus further degrading nearshore habitats for native fauna (Battelle et al. 2001).

Small intertidal areas of marsh and unvegetated marsh habitat in the LDW have become the focus of habitat restoration activities (www.darcnw.noaa.gov/eb.htm). The objectives of these projects include the removal of rock riprap and over-water wharf structures, restoration of natural tidal flow, and natural colonization by native wetland plants (Cordell et al. 1996).

A.2.1.3 Hydrologic data

The Green River, which is the main water source for the LDW, originates at the crest of the Cascade Mountains near Stampede Pass and flows through Howard Hanson Dam at 105 km (River Mile [RM] 65) and Tacoma Headworks Dam at 98 km (RM 61) (Culhane et al. 1995). Major tributaries to the Green River include Sunday Creek, Smay Creek, and the North Fork upstream of Howard Hanson Dam, and Newaukum Creek, Soos Creek, and Mill Creek downstream of Howard Hanson Dam. In addition to the Green River, the Black River continuously discharges fresh water to the LDW. These

flows are normally low (approx. 2.6 m^3/s [92 cfs]), but substantially increase from runoff during storms.

In the mid-1800s, discharge from the Duwamish ranged from an estimated 70 to 250 m^3/s (2,500 to 9,000 cfs) (Blomberg et al. 1988). The lower 10 km (6.2 mi) of the river was contained within a tidal marsh that opened into a broad expanse of intertidal flats. The Howard Hanson Dam was installed in the upper part of the Green River primarily for flood control and low-flow augmentation to preserve fish life when river flows were naturally low (Sato 1997).

Recent average discharge from the river was 43 to 51 m^3/s (2,300 to 2,350 cfs), measured at the USGS Tukwila gaging station, with flow rates varying from 4.3 to 329 m^3/s (200 to 15,200 cfs) (NOAA 1998). Most (80%) of the water flows out of the West Waterway due to the presence of a sill at the southern end of the East Waterway (Weston 1999). Flow rates are greatest in the winter due to seasonal precipitation and lowest throughout the late summer dry season. Streamflow can be increased by surface water sources such as storm drains, combined sewer overflows (CSOs), industrial effluents, and nonpoint inputs, although these sources of flow are expected to be less than 1% of total discharge.[9]

Flow has decreased 78% from historical levels, due mostly to the diversion of the White River to the Puyallup and the creation of the Ballard Locks and Cut. These changes lowered Lake Washington and caused increased drainage through the locks rather than through the Black River. Collectively, these irreversible changes have resulted in the present LDW hydrology and landscape.

Streamflow to the LDW is also influenced by water diversions, particularly by the City of Tacoma's Headworks Dam, which diverts at least 3.2 m^3/s (110 cfs) daily for municipal use. Discharge of effluent from the Renton Sewage Treatment Plant to the Duwamish River was eliminated in 1986, decreasing summer flows by as much as 25% (~1.6 m^3/s) (Harper Owes 1981; Bernhardt and Yake 1981).

A.2.1.4 Estuarine features

The LDW flows into Elliott Bay along the eastern shore of central Puget Sound. The LDW is a well-stratified salt-wedge type estuary that is influenced by river flow and tidal effects. Typical of salt-wedge estuaries, the Duwamish has a sharp interface between the freshwater outflow at the surface and saltwater inflow at depth. The 25-ppt layer of salt water near the river mouth occupies most of the water depth, but tapers towards the upriver portion of the estuary. The location where saltwater intrusion tapers to zero is called the toe of the salt wedge. In the LDW, the toe is located approximately 12 km (7.5 mi) upstream of the river mouth in the vicinity of Turning Basin 3. During summer low-flow conditions, the time of maximum salt

[9] Storm drain discharges to the LDW were estimated at 1,868 million gallons/year (MGY) (0.2 m^3/s) by Tetra Tech (1988) and CSO discharges are estimated at 20-25 MGY (0.002-0.003 m^3/s) in Tables 4-11 and 4-12 of the RI.

wedge incursion and tidal stage variation, the salt wedge toe can extend up to 16 km (RM 10) near Tukwila (Warner and Fritz 1995). Tidal effects and volume of river flow control movement of the salt wedge. At flow rates greater than 28 m^3/s, the wedge remains downstream of 12 km (RM 7.5) regardless of tidal stage. When flow rates are below 28 m^3/s, the lower 5.5 km of the estuary grades into a partially mixed estuary type (King County 1995). Dye studies indicate that downward vertical mixing over the length of the salt-wedge is almost nonexistent (Schock et al. 1998). Freshwater inflow and the occurrence of either ebb or flood tides within Elliott Bay heavily influence currents. Tides influence the entire length of the LDW. Upstream tidal flow reversal has been observed in the Green River 21 km (13 mi) upstream of the mouth. Additional information on temperature and salinity is presented in Sections 2.2.3 and 2.2.4 of the RI.

A.2.1.5 Sediment dynamics and load

Bottom sediment composition is variable throughout the LDW, ranging from sands to mud, depending on the sediment source and current speed. The sediment typically consists of slightly sandy silt with varying amounts of organic detritus. Previous data suggest coarser sediments are present in nearshore areas adjacent to CSO and storm drain discharges (Weston 1999). Finer-grain sediments are typically located in remnant mudflats, along channel sideslopes, and within portions of the navigation channel. Main channel sediments near the head of navigation are predominantly sands, whereas sediments toward the mouth are predominately fine-grained silts.

Roughly 99% of the total sediment load entering the LDW originates within the upstream Green River watershed (Harper-Owes 1983). Sediment loads measured at Renton Junction (19.3 km upstream; RM 12) have been shown to vary with streamflow; higher flows carry significantly greater amounts of material (Harper-Owes 1983). Measurements indicate bedload has at least historically been proportional to streamflow; bedload ranges from 20-40% of the suspended load (Stevens Thompson & Runyan 1972). Nearly 90% of the incoming sediment load to the LDW deposits within the dredged waterway reaches (Harper-Owes 1983). Significant export of sediment out of the LDW to the West Waterway and Elliott Bay only occurs during periods of high river discharge (greater than 200 m^3/s; 7,000 cfs) (Harper-Owes 1983; Curl et. al. 1987). Hydrodynamics within the LDW, specifically the location of the salt wedge, control the location of bedload deposition and shoaling within the waterway (Schultz and Tiffarny 1965). When fresh river water encounters the upstream end of the LDW salt wedge, the fresh water no longer applies a shear stress to the riverbed, but instead applies a stress to the top of the salt wedge. As the salt wedge is normally maintained in the vicinity of Turning Basin 3, bedload typically deposits within this area. Turning Basin 3 is specifically designed and managed to provide a settling basin for the bulk of the bedload sediment coming downstream from the undredged portions of the Duwamish River. The ACOE routinely dredges the area in the vicinity of Turning Basin 3 about every two years. Dredging records of the ACOE indicate the

total sediment volume transported into the LDW averages approximately 115,000 m^3/year (Grette and Salo 1986). Additional information regarding sediment transport is presented in Section 4.4 of the RI.

A.2.2 Resources Potentially at Risk

This section provides an overview of the ecological resources that utilize the LDW, including threatened, endangered, and sensitive species. These resources are considered in four groups, which include important species that could be directly or indirectly exposed to contaminated sediments: benthic invertebrates, fish, wildlife, and plants. Representative species from these groups are selected as ROCs, as discussed in Section A.2.3, and further evaluated to determine whether they may be at risk from contaminated sediments. Reptiles and amphibians are not likely to be exposed to sediment contamination in the LDW because habitat for these species is limited and their presence has not been reported in any wildlife surveys conducted in the area[10] (Canning et al. 1979; Cordell et al. 1996, 1997, 1999). Therefore, they will not be evaluated further in this ERA.

A.2.2.1 State and federal threatened, endangered, and sensitive species in the LDW

Fourteen species reported in the LDW are listed under either the federal Endangered Species Act (ESA) or by the Washington State Department of Fish and Wildlife as candidate species, threatened species, endangered species, or species of concern (Table A-2-1).

Eight of these fourteen species are fish and six are birds. With the exception of chinook salmon, coho salmon, bull trout, bald eagle, western grebe, and perhaps Pacific herring, use of the LDW by these species is rare or incidental, so they are not likely to have frequent exposure to sediment-associated chemicals from the LDW. Reports of these rare or incidental species in the LDW are from the following documents: loons (Canning et al. 1979, rare), merlin (Cordell et al. 1997, rare), common murre (believed to be rare), rockfish (Matsuda et al. 1968, rare; Malins et al. 1980, present), river lamprey (Warner and Fritz 1995, rare; Matsuda et al. 1968, rare), walleye pollock (Matsuda et al. 1968, rare; Miller et al. 1975, rare), and Pacific cod (Miller et al. 1975, 1977a; Weitcamp and Campbell 1980). Reports of peregrine falcon are anecdotal (Anderson 2002). These species share life history traits with other more common species in the LDW such that analysis of exposure and effects due to sediment-associated chemicals for the more common species should be protective of these species of concern. NMFS ruled on November 22, 2000 that listing of Pacific cod and walleye pollock under the ESA is not warranted (65FR227, Friday, November 24, 2000). NMFS ruled on April 3, 2001 that listing of Pacific herring, brown rockfish, copper rockfish, and quillback rockfish under the ESA is not warranted (66FR64, Docket No. 010312061-1061-01; I.D. 061199B]). Use of the LDW by chinook salmon,

[10] Note that a large tadpole was observed once in Slip 4.

coho salmon, bull trout, and herring is described in Section A.2.2.3. Use of the LDW by bald eagles and grebe is described in Section A.2.2.4.

Table A-2-1. Species listed under ESA or by Washington State Department of Fish and Wildlife

COMMON NAME	SCIENTIFIC NAME	STATUS
Chinook salmon	*Oncorhynchus tshawytscha*	FT, SC
Coho salmon	*Oncorhynchus kisutch*	FC
River lamprey	*Lampetra ayresi*	FSC, SC
Bull trout	*Salvelinus confluentes*	FT, SC
Pacific herring	*Clupea herengus pallasi*	SC
Pacific cod	*Gadus macrocephalus*	SC
Walleye pollock	*Theragra chalcogrammus*	SC
Rockfish species	*Sebastes* spp.	SC
Bald eagle	*Haliaeetus leucocephalus*	FT[a], ST
Peregrine falcon	*Falco peregrinus*	FSC, SS[b]
Merlin	*Falco columbarius*	SC
Common murre	*Uria aalge*	SC
Common loon	*Gavia Immer*	SS
Western grebe	*Aechmophorus occidentalis*	SC

Source – WDFW 2003

FT – Federal threatened species

FC – Federal candidate species

FSC – Federal species of concern

ST – State threatened species

SC – State candidate species

SS – State sensitive species

[a] Listing currently under review for removal

[b] Downlisted from state endangered to state sensitive April, 2002

A.2.2.2 Benthic invertebrates

Benthic invertebrate species are important components of the LDW ecosystem because they serve as a major food resource for commercially and recreationally important fish and wildlife, and because they are active in critical nutrient cycling. Benthic invertebrates in the LDW include 187 taxa, representing 46 families in 10 phyla (Table A-2-2). Typical of most estuaries, the invertebrate community is dominated by annelids, mollusks, and arthropods. Annelids are the most diverse of these three groups in the LDW, comprising 75 taxa of polychaete worms. The mollusks are represented by various bivalves and to a lesser extent by gastropods. Amphipods are the most diverse group of arthropods documented.

Table A-2-2. Species list of benthic invertebrates in the LDW

Phylum	Class	Order	Family	Genus and Species
Bryozoa				
Cnidaria				
	Hydrozoa			
		Hydroida		
	Anthozoa (sea anemones)			
		Actiniaria		
			Edwardsiidae	
				Edwardsia sp.
				Edwardsia californica
				Edwardsia callimorpha
				Edwardsia leidya
				Edwardsia sipunculoids
Platyhelminthes				
	Turbellaria (flatworms)			
		Polycladida		
			Stylochidae	
				Kaburakia excelsa
Nemertea (proboscis worms)				
	Anopla			
		Heteronemertea		
			Lineidae	
				Cerebratulus californiensis
				Cerebratulus sp.
		Palaeonemertea		
			Tubulanidae	
				Tubulanus sp.
	Enopla			
		Hoplonemertea		
Nematoda				
Annelida (segmented worms)				
	Archianellida			
	Oligochaeta			
			Megascolecidae	
				Enchytraeus sp.
			Naididae	
				Paranais sp.
	Polychaeta			
			Ampharetidae	
				Ampharete lobrops
				Amphicteis sp.
				Amphicteis scaphobranchiata
				Asabellides lineata
				Pseudoamphicteis sp.
				Hobsonia florida
			Arabellidae	
			Arenicolidae	
				Abarenicola pacifica

Phylum	Class	Order	Family	Genus and Species
			Capitellidae	
				Capitella capitata
				Heteromastus filiformis
				Heteromastus filobranchus
				Heteromastus sp.
				Mediomastus sp
				Nodomastus sp.
			Cirratulidae	
				Aphelochaeta sp.
				Aphelochaeta monilaris
				Chaetozone setosa
				Chaetozone sp.
				Cirratulus sp.
				Tharyx multifilis
			Cossuridae	
				Cossura sp.
				Cossura pygodactylata
			Dorvilleidae	
			Eunicidae	
			Glyceridae	
				Glycera americana
				Glycera nana
				Glycera capitata
			Goniadidae	
				Glycinde picta
				Glycinde polygnatha
				Goniada sp.
				Goniada maculate
			Hesionidae	
				Podarkeopsis glabra
			Lumbrineridae	
				Lumbrineris luti
				Scoletoma luti
			Maldanidae	
				Euclymene zonalis
				Euclymeninae sp.
			Nephtyidae	
				Nephtys sp.
				Nephtys comuta
				Nephtys ferruginea
			Nereidae	
				Neanthes sp.
				Nereis sp.
				Platyneris bicanaliculata
			Onuphidae	
				Onuphis iridescens
			Opheliidae	

Phylum	Class	Order	Family	Genus and Species
				Ammotrypane sp.
				Ammotrypane aulogaster
				Armandia brevis
				Ophelina acuminata
			Orbiniidae	
				Levinsenia gracilis
				Scoloplos sp.
			Paraonidae	
				Aricidea lopezi
			Pectinariidae	
				Pectinaria californiensis
			Phyllodocidae	
				Anaitides sp.
				Eteone longa
				Eteone sp.
				Phyllodoce sp.
			Pilargiidae	
				Pilargus maculata
			Polynoidae	
				Tenonia priops
			Sabellidae	
				Sabella sp.
				Manayunkia aestuarina
				Fabricia pacifica
				Fabricia sp.
			Sigalionidae	
				Pholoe sp.
				Pholoe minuta
			Sphaerodoridae	
				Sphaerodoropsis sphaerulifer
			Spionidae	
				Dipolydora caulleryi
				Laonice sp.
				Polydora uncata
				Polydora cornuta
				Polydora cardilia
				Polydora quadrilobata
				Polydora sp.
				Prionospio sp.
				Prionospio jubata
				Paraprionospio pinnata
				Pseudopolydora kempi japonica
				Pseudopolydora paucibranchiata
				Pygospio elegans
				Pygospio sp.
			Syllidae	
				Exogone lourei
			Terebellidae	
				Amphitrite cirrata
				Artacama coniferi
				Lanassa venusta venustai
				Polycirrus sp.
Mollusca				
	Bivalvia			
		Myoida		
			Hiatellidae	
				Hiatella arctica
			Myidae	
				Cryptomya californica
				Mya arenaria
		Mytiloida		
			Mytilidae	
				Megacrenella columbiana
				Mytilus edulis
		Nuculoidea		
			Nuculidae	
				Nucula tenuis
			Nuculanidae	
				Nuculana minuta
		Ostreoida		
			Anomiidae	
				Pododesmus cepio
		Pholadomyoida		
			Lyonsiidae	
				Lyonsia californica
			Pandoridae	
				Pandora filosa
				Pandora sp.
			Thraciidae	
				Thracia trapezoides
		Veneroida		
			Cardiidae	
				Clinocardium sp.
				Clinocardium nuttali
			Kelliidae	
				Odontogena *borealis*
			Lucinidae	
				Lucinoma acutlineata
				Parvilucina tenuisculpta
			Montacutidae	
				Mysella tumida
				Mysella sp.
			Solenidae	
				Solen sicarius
			Tellinidae	
				Macoma balthica
				Macoma carlottensis
				Macoma elimata

Phylum	Class	Order	Family	Genus and Species
				Macoma expansa
				Macoma incongrua
				Macoma nasuta
				Macoma yoldiformis
				Macoma sp.
				Tellina sp.
			Thyasirdae	
				Axinopsida serricata
			Veneridae	
				Psephidia lordii
				Saxidomus giganteus
				Transennella tantilla
	Gastropoda (snails)			
		Mesogastropoda		
			Epitoniidae	
				Epitonium sp.
			Melanellidae	
				Melanella sp.
			Rissoidae	
				Alvania compacta
				Barleeia sp.
			Turritellidae	
				Tachyrhynchus sp.
		Neogastropoda		
			Nassinae	
				Nassarius sp.
			Columbellidae	
				Alia carinata
				Mitrella gouldii
				Nitidella gouldi
	Opisthobranchia (subclass)			
			Pyramidellidae	
				Odostomia sp.
		Nudibranchia		
			Aeolidacea	
		Cephalaspidea		
			Gastropteridae	
				Gastropteron pacificum
			Doridiidae	
				Melanochlamys diomedea
		Pteropoda		
	Aplacaphora			
			Chaetodermatidae	
				Chaetoderma sp.
Arthropoda				
	Arachnida			
		Acari		
			Halacaridae	

Phylum	Class	Order	Family	Genus and Species
	Crustacea			
		Amphipoda		
				Tritella pilimana
				Incisocalliope sp.
				Eochelidium miraculum
				Chromopleustes oculatus
			Aoridae	
				Aoroides sp.
			Ampithoidae	
				Ampithoe sp.
			Anisogammaridae	
				Eogammarus confervicolus
				Anisogammarus confervicolus
				Anisogammarus sp.
			Caprellidae	
			Corophiidae	
				Corophium acherrusicum
				Corophium salmonis
				Corophium spinicorne
				Corophium insidiosum
				Corophium sp.
			Eusiridae	
				Paramoera sp.
			Ischyroceridae	
				Protomedeia sp.
			Melitidae	
				Melita desdichada
			Oedicerotidae	
				Americhelidium shoemakeri
				Monoculoides sp.
				Westwoodilla caecula
			Podoceridae	
				Dyopedos sp.
		Cladocera		
			Podonidae	
				Podon leuckarti
		Euphausiacea		
			Euphausid	
		Isopoda		
			Paramunnidae	
				Munnogonium sp.
				Munnogonium tillerae
			Pleurogoniidae	
				Pleurogonium rubricundum
			Sphaeromatidae	
				Gnorimosphaeroma oregonesis
			Epicaridea	
		Cumacea		
			Diastylidae	

Phylum	Class	Order	Family	Genus and Species
				Diastylis santamariensis
			Lampropidae	
				Lamprops quadriplicata
			Nannastacidae	
				Cumella vulgaris
			Leuconidae	
				Eudorella pacifica
				Nippoleucon hinumensis
		Tanaidacea		
				Leptochelia sp.
				Leptochelia savignyi
				Sinelobus stanfordi
				Tanais sp.
		Mysidacea		
			Mysidae	
				Neomysis mercedis
				Alienacanthomysis macropsis
		Decapoda		
			Cancridae	
				Cancer oregonensis
			Crangonidae	
				Crangon sp.
				Crangon alaskensis
			Hippolytidae	
				Eualus pusiolus
			Pinnotheridae	
				Pinnixa schmitti
		Thoracica		
			Balanomorpha (suborder)	
			Balanidae	
				Balanus crenatus
	Copepoda (subclass)			
		Harpacticoida		
			Ancorabolidae	
			Ameiridae	
				Ameira sp.
				Nitocra sp.
			Canthocamptidae	
				Leimia vaga
				Cletocamptus sp.
				Mesochra sp.
				Mesochra rapines
			Canuellidae	
				Coullana canadensis
			Cletodidae	
				Acrenhydrosoma sp.
				Enhydrosoma sp.
			Cylindropsyllidae	
			Darcythompsoniidae	

Phylum	Class	Order	Family	Genus and Species
			Diosaccidae	
				Amphiascopsis cinctus
				Amphiascopsis sp.
				Amphiascoides sp.
				Amonardia perturbata
				Amonardia normani
				Diosaccus sp.
				Diosaccus spinatus
				Bulbamphiascus sp.
				Robertsonia sp.
				Typhlamphiascus pectinifer
				Typhlamphiascus sp.
				Stenhelia asetosa
				Stenhelia peniculata
				Stenhelia sp.
				Schizopera knabi
				Schizopera sp.
			Ectinosomatidae	
				Pseudobradya sp.
			Harpacticidae	
				Harpacticus uniremis
				Harpacticus sp.
				Harpacticus compressus
				Harpacticus obscurus
				Harpacticus spinulosus
				Harpacticus arcticus
				Zaus sp.
			Huntemanniidea	
				Nannopus palustris
				Huntemannia jadensis
			Laophontidae	
				Heterolaophonte discophora
				Heterolaophonte longisetigera
				Heterolaophonte hamondi
				Laophonte sp.
				Laophonte cornuta
				Laophonte elongata
				Echinolaophontes sp.
				Onychocamptus mohammed
				Paralaophonte sp.
				Paralaophonte pacifica
				Paralaophonte perplexa
				Pseudonychocamptus sp.
			Longipediidae	
				Longipedia sp.
			Normanellidae	
				Normanella sp.
			Orthopsyllidae	
				Orthopsyllus illgi

Phylum	Class	Order	Family	Genus and Species
			Paramesochridae	
				Apodopsyllus sp.
			Parastenheliidae	
				Parastenhelia hornelli
				Parastenhelia spinosa
			Peltidiidae	
			Tachidiidae	
				Microarthridion littorale
				Tachidius disciples
				Tachidius triangularis
			Tegastidae	
			Thalestridae	
				Dactylopodia crassipes
				Dactylopodia vulgaris
				Dactylopodia tisboides
				Dactylopodia paratisboides
				Dactylopodia glacialis
				Diarthrodes sp.
				Idomene sp.
				Paradactylopodia sp.
				Parathalestris sp.
				Rhynchothalestris helgolandica
			Tisbidae	
				Scutellidium sp.
				Tisbe sp.
		Cyclopoida		
			Cyclopoidae	
				Halicyclops sp.
			Oithonidae	
				Oithona similis
				Oithona longirastris
		Calanoida		
			Temoridae	
				Eurytemora sp.
				Eurytemora americana
			Centropagidae	
				Centropages abdominalis
			Pseudodiaptomidae	
				Pseudodiaptomus marinus
			Stephidae	
				Stephos sp.
			Calanidae	
				Calanus sp.
			Paracalanidae	
				Paracalanidae sp.
			Clausocalanidae	
				Microcalanus sp.
				Pseudocalanus sp.
			Acartiidae	
				Acartia sp.
				Acartia longiremis
		Poecilostomatoida		
			Corycaeidae	
				Corycaeus anglicus
			Clausidiidae	
				Hemicyclops sp.
			Ergasilidae	
			Oncaeidae	
				Oncaea sp.
	Ostracoda			
		Myodocopida		
			Cylindroleberididae	
			Philomedidae	
				Euphilomedes carcharodonta
		Podocopida		
	Insecta (larvae)			
		Ceratopogonidae		
		Coleoptera		
		Diptera (pupa)		
			Dolichopodidae (larvae)	
			Chironomidae (larvae)	
		Empididae		
		Collembola		
		Trichoptera		
		Thysanoptera		
Echinodermata				
	Stelleroidea			
		Ophiurida		
			Amphiuridae	
				Amphiodia sp.
				Amphiodia digitata
	Holothuroidea			
		Dendrochirotida		
			Cucumariidae	
				Pentamera sp.
Cephalorhyncha				
	Priapulida			
			Priapuloidae	
				Priapulus caudatus
Rhizopoda				
	Rhizopodea			
		Foraminiferida		
Rotifera				

Sources: Bingham (1978); Leon (1980); Williams (1990); Cordell et al. (1996, 1997); Taylor et al. (1999); Striplin (1998)

Average abundances of various invertebrate groups were reported in King County (1990), Leon (1980), and Williams (1991). The average abundance of many of the larger invertebrate species captured in Puget Sound Ambient Monitoring Program (PSAMP) otter trawls is presented in Table A-2-3.

Table A-2-3. Average abundance per trawl of invertebrate species collected in PSAMP otter trawls from LDW stations[a]

Name	Average Abundance per Trawl
Graceful crab	16.7
Crangonid shrimp unidentified	11.5
Gigantic anemone	6.2
False ochre star	3.8
Dungeness crab	2.5
Coonstriped shrimp	2.3
Pink short spined seastar	1.8
Dock shrimp	0.8
California arminid	0.7
Basket cockle	0.5
Leather star	0.3
Porcelain crab	0.3
Sunflower star	0.3
Oregon cancer crab	0.2
Chiton (unidentified)	0.2
Rose sea star	0.2
Scarlet anemone	0.2

Source: West 2001

[a] A total of six otter trawls were conducted on 5/18/1992, 5/29/1992, 5/4/1995, 4/14/1997 (3 trawls) at depths of 5.5 to 11 m near Kellogg Island.

Dungeness and several other crab species are found in the LDW; their distribution is generally limited to the lower part of the estuary where salinity is greater. During fieldwork conducted by Environmental Solutions Group (ESG 1999), adult Dungeness and red rock crabs were collected at multiple locations near Kellogg Island, but could not be caught upstream of this point. Juvenile Dungeness crabs were found up to the 1st Avenue South Bridge.

Estuarine use by Dungeness crabs is dependent on their life stage, as described below based on information presented in Pauley et al. (1986). Dungeness crabs usually mate in offshore locations, but occasionally mate in estuaries. Spawning takes place offshore. Eggs mature in about 2-3 months, and hatch in January through April in Washington. Larvae appear to be transported seaward from the onset of hatching with a distribution dependent on depth, latitude, temperature, salinity, and currents.

Larvae progress through five zoeal states before molting into megalopae. Megalopae first appear in April in Washington waters, with abundance peaking in May through June, after which they molt into juveniles. The juveniles are found in shallow coastal waters and estuaries, like the LDW, and large numbers can live among eelgrass or other aquatic vegetation.[11] Subadult and adult Dungeness crab tend to live offshore, whereas juveniles are more common in estuaries. Most adult female crabs tagged off the coast of Northern California moved relatively little (about 1.6 km). Along the coast of southern Washington, legal-size males (about 4 years old) generally moved inshore and toward the estuaries in the fall.

The diet of crabs is dependent on their life stage (Pauley et al. 1986). Larvae eat both zooplankton and phytoplankton. The diet of juvenile crabs consists largely of fish, mollusks, and crustaceans. Adult crabs prey on clams, crustaceans, and fish. Crabs progress from eating bivalves their first year, to eating shrimp their second year, to eating teleost fish their third year. Megalopae are preyed upon by many fishes including salmon. Juvenile crabs are preyed upon by various demersal fishes in the nearshore area with flatfishes, such as starry flounder and English sole, being the most important predators. Adult and juvenile crabs are preyed upon by sea otters, fishes, and octopuses. Cannibalism is also common among crabs.

Bivalves are also common in the LDW. Windward Environmental (2000) conducted a reconnaissance survey to document the presence or absence of bivalves in the intertidal zone of several areas[12] within the LDW. This survey was an initial effort to understand more about the abundance and distribution of clams. Samples were collected by shovel using randomly placed transects and directed sampling. Only one clam was found using randomly placed transects; most of the clams were found when siphon holes in probable places were investigated. Abundance was highest at Kellogg Island, but one or more clams were found at each sampling site. Five different species were identified: softshell clam (*Mya arenaria*), butter clam (*Saxidomus giganteus*), sand clam (*Macoma secta*), bent-nose clam (*Macoma nasuta*), and the inconspicuous macoma (*Macoma inconspicua*). Mussels were also observed in large numbers on pilings and other structures in the lower, more saline end of the LDW, although they have also been reported to occur up to and slightly above Turning Basin 3 in the LDW. Bivalves are siphon feeders obtaining their food either from the water column or the sediment surface, depending on their location.

The invertebrates present in the LDW are characterized as either infaunal or epibenthic. The infaunal community is typified by burrowing polychaetes and bivalves. This community is dominated by deposit- and filter-feeding organisms. King County (1999c) found that at the majority of stations sampled, the infaunal community was dominated by surface detrital- and surface deposit-feeding organisms. Larger

[11] No eelgrass is found in the LDW, and habitats with aquatic vegetation are rare (Battelle et al. 2001).
[12] Terminal 105, Kellogg Island, Slip 2, Slip 4, and Duwamish Yacht Club

crustaceans and mussels characterize the epibenthic community, which is also dominated by surface detrital and surface filter-feeding organisms.

In general, the key physical factors influencing benthic invertebrate species distribution and abundance are salinity, water depth, percent fine-grained sediment (i.e., silt and clay), salinity, and organic carbon content. Most benthos in the LDW are small (<0.5 mm), numerous, and found in fine-grained sediments (Cordell et al. 1999).

The LDW provides two distinct benthic habitats: intertidal habitat (frequently exposed by low tides) and subtidal habitat (rarely or never exposed by low tides). Species surveys of these two habitats are discussed in more detail in Sections A.2.2.2.1 and A.2.2.2.2. In many places in the LDW, intertidal habitat is present in shallow shelves, such as mudflats. Intertidal areas are often separated from the subtidal navigation channel in the center of the LDW by subtidal transition areas with steep slopes. Sediment characteristics with each of the three regions (i.e., shallow shelf, steep-sloped transition areas, and flat-bottomed navigation channel) may be different.

A.2.2.2.1 Intertidal

Intertidal habitats include mudflats, sandflats, and hard surfaces. The majority of the LDW shoreline is composed of riprap, pier aprons, or sheet piling (Tanner 1991). These hard surfaces support populations of encrusting organisms such as barnacles and burrowing organisms such as shipworms (Leon 1980). Remnants of natural intertidal habitat occur on the northern portion of Kellogg Island and in occasional patches throughout the LDW.

Leon (1980) found 43 different benthic taxa in sediment cores from the intertidal mudflats at Kellogg Island. Most organisms occurred infrequently; nine taxa accounted for 97% of all individuals. Small marine worms of the genus *Manayunkia*, oligochaetes, and harpacticoid copepods made up nearly 80% of all individuals (Leon 1980). In comparison, there were very few organisms at a mudflat site with anoxic sediments near the Duwamish Shipyards, and there was a greater degree of seasonal variability in the benthic community at a mudflat site in the marina near Kellogg Island.

Williams (1990) identified 80 invertebrate taxa inhabiting intertidal habitats at Kellogg Island. Nematodes, oligochaetes, small harpacticoid copepods, ostracods, and sabellid polychaetes were the dominant forms. Cordell et al. (1999) presented results of a 1997 monitoring study in the LDW that included an evaluation of benthic macrofauna (>0.5 mm) and meiofauna (0.045-0.5 mm) collected with core tubes from four sampling sites distributed throughout the LDW. Abundant macrofauna included species of Oligochaeta, Polychaeta, and Amphipoda. Abundant meiofauna included species of harpacticoid copepods, Nematoda, Foraminifera, and Ostracoda.

Cordell et al. (1999) conducted epibenthic and infaunal surveys at seven restoration and reference sites throughout the LDW from 1993 through 1997. They found diversity and abundance of intertidal organisms varied seasonally and among locations in the

LDW. The greatest diversity of organisms (i.e., species richness) occurred in the lower LDW; diversity was comparatively lower in Turning Basin 3. Seasonally, species diversity and abundance increased from winter through summer as primary productivity increased. In spring, community composition was generally dominated by two to three species. By summer, the species composition was generally more evenly distributed among a greater number of species. At all sites sampled, the macrofauna were generally numerically dominated by oligochaetes, polychaete worms of the genus *Manayunkia*, and gammarid amphipods of the genus *Corophium*. The meiofauna at all sites sampled were generally dominated by nematodes and harpacticoid copepods. The authors attribute the differences in diversity and abundance among sites to differences in sediment grain size, intertidal vegetation, disturbance from boat traffic and dredging, and to greater fluctuations in salinity at the upstream sites.

Many animals that live in the intertidal zone, particularly polychaete worms such as *Capitella* sp., feed on particles of decaying plant and animal matter deposited on the sediments. Other organisms, such as copepods, feed on diatoms, detritus, and larvae. Oligochaetes feed on bacteria, diatoms, and other microorganisms (King County 1999c). However, some benthic invertebrates, such as the polychaete *Manayunkia* sp., filter particles from the water column.

A.2.2.2.2 Subtidal

Subtidal habitat occurs throughout the LDW. Near Kellogg Island, the subtidal habitat is characterized as a brown or brown-gray sandy mud overlying darker, more clayey mud. Leon (1980) used van Veen grab samplers to characterize the epibenthic and infaunal sediment biota from near Kellogg Island. They found more than 60 different taxa, greater than the number found in the intertidal habitat from the same survey. The most abundant taxon was deposit-feeding cirratulid polychaete worms. While some of these subtidal animals were also found intertidally (oligochaetes, *Capitella* sp., *Pygospio* sp., ostracods), most subtidal species were deposit-feeding polychaete worms that are characteristic of the deeper, turbid waters of the LDW. Small deposit-feeding clams (*Macoma* sp., *Axinopsida* sp., and *Psephidia* sp.) and the amphipod *Anisogammarus* sp., which feeds on diatoms and green algae, were also present.

Parametrix (Williams 1990) sampled epibenthic sediment biota near Kellogg Island and found that nematodes, oligochaetes, small harpacticoids, and cumaceans dominated the subtidal epibenthos. As with the intertidal benthos, stations with finer sediments generally had a greater abundance of epibenthic biota.

Striplin (1998) evaluated risks to benthic infauna and epibenthos as a component of their assessment of CSO discharges to the Duwamish River and Elliott Bay. Subtidal samples were collected with a 0.1-m^2 van Veen grab sampler and organisms were retained using a 1.0-mm mesh sieve. Sampling sites included transects located at Kellogg Island and downgradient from the Duwamish/Diagonal CSO. Polychaeta were abundant in all samples and were the dominant organisms at all locations except

at two stations downstream of the Duwamish/Diagonal CSO, at which Oligochaeta and Mollusca were dominant. A Kellogg Island station also had relatively abundant Mollusca. Arthropoda tended to be more abundant in deeper waters.

In summary, invertebrates in the LDW consist of infauna and epibenthic organisms in both intertidal and subtidal zones. Larger species, such as mollusks, arthropods, and echinoderms, are also present. Although salinity varies throughout the site both temporally and spatially, saline waters reach the entire extent of the LDW, contributing to the diversity and abundance of species present.

A.2.2.3 Fish

The LDW is home to numerous anadromous and resident fish species. These species are listed in Table A-2-4, along with key information and citations. LDW fish studies are further summarized in Section 2.4.3.1 and Table 2-9 of the RI. Note that several of the LDW fish studies are more than ten years old; since these studies were conducted, the fish community may have changed as a result of changing conditions, such as habitat restoration.

Warner and Fritz (1995) recorded 33 species of resident and seasonal species of fish in the LDW. Miller et al. (1975, 1977a) observed a total of 29 species and Matsuda et al. (1968) recorded a total of 28 species. In these studies, shiner surfperch, snake prickleback, Pacific sandlance, Pacific staghorn sculpin, longfin smelt, English sole, and starry flounder were particularly abundant, as were chinook, chum, and coho salmon. This section discusses the most abundant fish that utilize the LDW. Fish abundance peaks in late summer to early fall and is generally lowest in winter (Miller et al. 1977a; Dexter 1981). Based on otter trawl data, species richness was shown to follow a similar trend but did not vary greatly with season (Miller et al. 1977a).

Table A-2-4. Fish species in the LDW

Common Name	Scientific Name	Family	Abundance	Citation	Environment	Habitat	Citation	Diet	Citation
Bay goby	*Lepidogobius lepidus*	Gobiidae	r	2, 3, 6	marine (estuary)	benthic (mud bottom)	9	benthic organisms	25
Bay pipefish	*Syngnathus grisiolineatum*	Syngnathidae	r	6	marine	demersal (associated with eel grass in the intertidal areas)	11	isopods, amphipods	10
Big skate	*Raja binoculata*	Rajidae	r	7	marine	benthic (sandy and gravelly bottoms)	12	crustaceans, fish	10
Buffalo sculpin	*Enophrys bison*	Cottidae	r	1, 2, 3, 4, 7	marine (estuary)	benthic (inshore rocky and sandy areas)	9	mainly algae, also amphipods, small fishes, crabs, polychaetes, nudibranchs, isopods	9, 26
Bull trout	*Salvelinus confluentes*	Salmonidae	r	6	anadromous	benthopelagic (near shore)	17	mainly fish, plus zooplankton	28
Butter sole	*Isopsetta isolepis*	Pleuronectidae	c, (r)	6, (7)	marine (estuary)	benthic (sandy bottom)	9	worms, fish, shrimps	10
Chinook salmon [a]	*Oncorhynchus tshawytscha*	Salmonidae	a, (r)	1, 4, 5, 6, (2)	anadromous	benthopelagic	24	juveniles: insects, epibenthic crustaceans, pelagic organisms	27
Chum salmon	*Oncorhynchus keta*	Salmonidae	r (a)	1, 4, (5, 6)	anadromous	benthopelagic	24	juveniles: copepods, amphipods, cumaceans, euphausiids	26
C-O sole	*Pleuronichthys coenosus*	Pleuronectidae	r	7	marine	benthic (flat bottoms, rocky areas)	9	isopods, fish, polychaetes, amphipods, turbellarians, bivalves	26
Coho salmon [a]	*Oncorhynchus kisutch*	Salmonidae	r, (c), [a]	1, 2, (4), [6]	anadromous	benthopelagic	24	juveniles: insects, epibenthic crustaceans, pelagic organisms, small fish	26
Cresent gunnel	*Pholis laeta*	Pholidae	r	6	marine (estuary)	demersal (intertidal areas, under rocks)	9	gammarid amphipods, copepods, tanaids, isopods	26
Cutthroat trout	*Oncorhynchus clarki*	Salmonidae	r	1, 4, 5, 6	anadromous	benthopelagic	18	fish, epibenthic crustaceans, pelagic organisms, insects	14
Dolly Varden	*Salvelinus malma*	Salmonidae	r	1, 4	fresh water	benthopelagic	17	fish, epibenthic crustaceans, pelagic organisms, insects	10
Dover sole	*Microstomus pacificus*	Pleuronectidae	c, (r)	2, (3)	marine	benthic (mud bottom)	9	benthic invertebrates, echinoderms, mollusks, polychaetes	20

Common Name	Scientific Name	Family	Abundance	Citation	Environment	Habitat	Citation	Diet	Citation
English sole	*Parophrys vetulus*	Pleuronectidae	a, (r)	2, 3, 4, 7 (1,6)	marine (estuary)	benthic (sand and mud bottoms)	14	cumaceans, gammarid amphipods, polychaetes, tanaids, crabs, bivalves	26
Eulachon	*Thaleichthys pacificus*	Osmeridae	i	3	anadromous	pelagic	9	plankton (only feeds while at sea)	16
Flathead sole	*Hippoglossoides elassodon*	Pleuronectidae	i	2	marine	benthic (soft mud bottom, adults below 180m)	9	polychaetes, cumaceans, gammarid amphipods, isopods, bivalves	26
Hybrid sole	*Inopsetta Isopsetta ischyra*	Pleuronectidae	r	1	marine (estuary)	benthic	9	benthic organisms	10
Largescale sucker	*Catostomus macrocheilus*	Catostomidae	i (r)	1, 2, 4, (6)	fresh water	demersal	17	algae, diatoms, insects, amphipods, and mollusks	16
Longfin smelt	*Spirinchus thaleichthys*	Osmeridae	a, (r)	1, 2, (7)	anadromous	benthopelagic (close to shore, in bays and estuaries)	17	crab larvae, copepods, mysid shrimp	26
Longnose dace	*Rhinichthys cataractae*	Cyprinidae	i	6	fresh water	demersal	17	mayflies, blackflies, and midges	16
Mountain whitefish	*Prosopium williamsoni*	Salmonidae	i	1, 6	fresh water	benthopelagic	10	insects, inverts, eggs, small fish	10
Northern pikeminnow	*Ptychocheilus oregonensus*	Cyprinidae	i	1, 6	fresh water	benthopelagic	16	insects, fish	16
Northern sculpin	*Icelinus borealis*	Cottidae	r	6	marine	demersal	9	benthic crustaceans, shrimps/prawns	10, 26
Pacific cod	*Gadus macrocephalus*	Gadidae	r	2, 3, 4	marine	(demersal, continental shelf and upper slopes)	19	fish, octopi, large crustaceans, worms, amphipods	22, 26
Pacific herring	*Clupea pallasi*	Clupeidae	c, (a), [r]	1, 2, 7, (4), [6]	marine	benthopelagic (coastal, 1st yr in bays)	10	planktonic crustaceans, fish larvae	10, 26
Pacific sandlance	*Ammodytes hexapterus*	Ammodytidae	c, (r), [a]	4, (1), [6]	marine (brackish)	benthopelagic (surface or burrowed in sand)	9	zooplankton	13, 26
Pacific staghorn sculpin	*Leptocottus armatus*	Cottidae	a, (c)	1, 2, 3, 4, 6, (7)	marine (lower estuary, offshore)	benthic (sandy bottom)	9	isopods, bivalve siphons, polychaetes, crabs, fish, tanaids, shrimp	15
Pacific tomcod	*Microgadus proximus*	Gadidae	r, (c), [a juvi]	1, 4, (2, 3), [7]	marine (brackish)	benthic (over sand)	19	shrimps, amphipods, isopods, gastropods, mussels, fishes	20

Common Name	Scientific Name	Family	Abundance	Citation	Environment	Habitat	Citation	Diet	Citation
Padded sculpin	*Artedius fennestralis*	Cottidae	c, (r)	2, 3, (7)	marine	benthic	9	gammarid amphipods, isopods, tanaids, shrimp, copepods, small fish	14, 26
Penpoint gunnel	*Apodichthys flavidus*	Pholidae	r	5, 6	marine (estuary)	demersal (intertidal-tidepools)	9	isopods, amphipods, shrimp, gastropods, other epibenthic crustaceans	26
Pile perch	*Rhacochilus vacca*	Embiotocidae	r, (c)	1, 2, 3, 6, (4, 7)	marine	demersal (rocky shores; near kelp, pilings, underwater structures)	9	isopods, bivalves, crabs, amphipods	26
Pink salmon [a]	*Oncorhynchus gorbuscha*	Salmonidae	r	6	anadromous	benthopelagic	24	juveniles: copepods, amphipods, barnacle larvae, cumaceans	2325
Plainfin midshipman	*Porichthys notatus*	Batrachoididae	i	2	marine	benthic (nearshore shelf, sand/mud bottom)	14	crustaceans, fish	10
Prickly sculpin	*Cottus asper*	Cottidae	r	1, 2, 3, 4, 6	marine	benthic	9	benthic organisms	16
Pygmy poacher	*Odontopyxis trispinosa*	Agonidae	i, (r)	2, 3, (7)	marine	demersal (soft bottoms)	9	epibenthic invertebrates	10
Ratfish	*Hydrolagus colliei*	Chimeridae	r	2, 7	marine	demersal (sandy bottom)	9	worms, bivalves, crustaceans, fishes	13, 26
Redsided shiner	*Richardsonius balteatus*	Cyprinidae	c	6	fresh water	demersal	16	zooplankton, algae, insects	16
River lamprey	*Lampetra ayresi*	Petromyzontidae	r	1, 4, 6	anadromous	demersal	10	adult: fish juveniles: detritus, algae	16
Rock sole	*Lepidopsetta bilineata*	Pleuronectidae	c, (a)	2, 3, (7)	marine (estuary)	benthic (more pebbly bottom than most other flatfish)	9	isopods, gammarid amphipods, polychaetes, cumaceans, bivalves, crabs, fish	26
Rockfish	*Sebastes* spp.	Scorpaenidae	r	1, 8	marine	demersal (near structure)	21	crabs, gammarid amphipods, mysids, shrimp, fish	22
Roughback sculpin	*Chitonotus pugeteneis*	Cottidae	i, (r)	2, (3, 7)	marine	benthic (sand/mud bottom)	9	shrimps and other crustaceans	14
Saddleback gunnel	*Pholis ornata*	Pholidae	r	3, 5, 6	marine (estuary)	demersal (sandy bottom)	9	amphipods, isopods, polychaete, copepods, cumaceans	26

Common Name	Scientific Name	Family	Abundance	Citation	Environment	Habitat	Citation	Diet	Citation
Sand sole	*Psettichthys melanostictus*	Pleuronectidae	c, (r)	1, 2, 3, 7, (1)	marine, estuary	benthic (sandy bottom)	10	fishes, worms, crustaceans and mollusks	10, 26
Sharpnose sculpin	*Clinocottus acuticeps*	Cottidae	i	6	marine	benthic (sand/vegetation)	9	benthic organisms	18
Shiner surfperch	*Cymatogaster aggregata*	Embiotocidae	a, (c)	1, 4, 5, 6, 7, (2, 3)	marine (estuary)	demersal (in shallow water, around eelgrass beds, piers and pilings commonly in bays and quiet back waters)	9	amphipods, cumaceans, polychaetes, copepods, isopods, algae	18, 26
Slender sole	*Lyopsetta exilis*	Pleuronectidae	i	3	marine	benthic (>200m depth)	9	carnivore	20
Snake prickleback	*Lumpenus saggita*	Stichaeidae	a, (r)	1, 2, 3, 4, 6, (7)	marine	benthopelagic (shallow bays and offshore waters)	9	bivalves, marine worms, amphipods	26
Sockeye salmon [a]	*Oncorhynchus nerka*	Salmonidae	i		anadromous	benthopelagic	24	juveniles: insects, epibenthic crustaceans, pelagic organisms	25
Soft sculpin	*Gilbertidia sigalutes*	Cottidae	r	4	marine	demersal	9	epibenthic crustaceans, phytoplankton, fish eggs/larvae	10
Speckled sanddab	*Citharichthys stigmaeus*	Bothidae	r	7	marine	benthic (sandy bottom)	9	crustaceans, fish	15
Spiny dogfish	*Squalus acanthias*	Squalidae	i	2	marine	benthopelagic	22	primarily fish	24
Starry flounder	*Platichthys stellatus*	Pleuronectidae	a, (c)	1, 2, 3, 4, 6, 7, (5)	marine (estuary, brackish)	benthic	18	isopods, fish, gammarid amphipods, polychaetes, gastropods, worms	10
Steelhead [a]	*Oncorhynchus mykiss*	Salmonidae	r	1, 4, 5, 6	anadromous	benthopelagic		juveniles: insects, epibenthic crustaceans, pelagic organisms	26
Striped seaperch	*Embiotoca lateralis*	Embiotocidae	r, (c)	2, 3, 5, 6, 7, (1, 4)	marine	demersal	9	amphipods, isopods, crabs, shrimp	26
Sturgeon poacher	*Podothecus acipenserinus*	Agonidae	i	3	marine	demersal (soft bottom)	9	cumaceans, gammarid amphipods, shrimp, copepods, polychaetes, tanaids	26
Surf smelt	*Hypomesus pretiosus*	Osmeridae	c	1, 4, 6, 7	marine (brackish)	benthopelagic	18	isopods, cumaceans, larvaceans, copepods, amphipods	26

Common Name	Scientific Name	Family	Abundance	Citation	Environment	Habitat	Citation	Diet	Citation
Three-spine stickleback	*Gasterosteus aculeatus*	Gasterosteidae	c, (r)	1, 5, 6 (4)	marine, anadromous	benthopelagic (in/near vegetation)	17	worms, crustaceans, insects/larvae, small fish	16, 26
Tubesnout poacher	*Pallasina barbata*	Agonidae	i	3	marine	demersal (eelgrass & seaweeds)	9	amphipods, polychaetes, copepods, mysids	26
Walleye pollock	*Theragra chalcogramma*	Gadidae	r	1, 2, 4	fresh water	benthopelagic	19	insects, midge larvae, fish	10
Whitespotted greenling	*Hexagrammos stelleri*	Hexagrammidae	i, (c)	2, (7)	marine (intertidal)	demersal (nearshore, near rocks, pilings and eelgrass beds)	19	gammarid amphipods, shrimp, crabs, fish, polychaetes	26

[a] Adults are found in the LDW only as they migrate to spawning ground upstream of the LDW

Abundance: a-abundant (numerically dominant), c-common (occurs in most samples), r-rare (occurs regularly in a few samples), i-incidental (not usually found in LDW). Letters in parentheses relate distinct abundance classification to citation; numbers in parentheses indicate the source of the distinct data. Abundance characterizations reflect data collected by authors in the cited study. These data may reflect sampling gear bias for the species identified.

Abundance citations: 1-Matsuda et al. (1968), 2-Miller et al. (1975), 3-Miller et al. (1977a), 4-Weitkamp and Campbell (1980), 5-Taylor et al. (1999), 6-Warner and Fritz (1995), 7–West et al. (2001); 8–Malins et al. (1980)

Biology citations: 9-Eschmeyer et al. (1983), 10-Hart (1973), 11-Dawson (1985), 12-McEachran and Dunn (1998), 13-Armstrong (1996), 14-Clemens and Wilbey (1961), 15-Fitch and Lavenberg (1975), 16-Scott and Crossman (1973), 17-Page and Burr (1991), 18-Morrow (1980), 19-Cohen et al. (1990), 20-Pearcy and Hancock (1978), 21-Lamb and Edgel (1986), 22-Cox and Francis (1997), 23- 24-Groot and Margolis (1991), 25-Grossman (1979), 26 Miller et al. (1977b), 27-Cordell et al. (2001), 28-Rieman and McIntyre (1993)

A.2.2.3.1 Anadromous salmonids – Pacific salmon

All species of Pacific salmon (coho, chinook, chum, sockeye, and pink) have been found in the LDW (King County et al. 2000). These anadromous fish use the estuary for rearing and as a migration corridor for adults and juveniles. Among numerous beneficial uses of the LDW identified by METRO, use as habitat for outmigrating juvenile salmonids was listed as the most important (Harper-Owes 1983). Salmonid residence time in the LDW depends on the specific life history characteristics of the species. Salmon found in the LDW spawn mainly in the middle reaches of the Green River and its tributaries (Grette and Salo 1986).

Timing of upstream migration of chinook, chum, and coho salmon is largely controlled by rainfall, streamflow, and barometric pressure (Ecology 2000). Adult salmon generally do not feed to any significant extent once they enter the river on their spawning migrations. The peak timing of outmigration for juveniles of all species generally corresponds with March-June high flows. Outmigration usually lasts through mid-July to early August for most species (Warner and Fritz 1995). During this time, juveniles use the estuary to feed and begin their physiological adaptation to higher salinity.

Chinook salmon

Historically, the Green/Duwamish River supported spring and fall runs of chinook salmon. Fall-run chinook are the only naturally sustaining run that still uses the Green/Duwamish River corridor. These chinook are a sub-population of the Puget Sound chinook population, which was listed as a threatened species under ESA in March 1999. These fish use the LDW for migration to and from spawning grounds in the mainstem Green River and larger tributary streams. Production is from hatcheries, naturally spawning hatchery-reared fish, and naturally spawning native fish (Grette and Salo 1986; WDFW 1993).

Returning fall chinook salmon enter the LDW from late June through mid November, with peak migration in mid August (Grette and Salo 1986). These fish spawn in the upper watershed from August through November, with a peak at the beginning of October (Becker 1967; Miller and Stauffer 1967; Williams et al. 1975, as cited in Grette and Salo 1986). Adult fish hold in the lower river from the mouth of the LDW to Kent until temperature conditions and sufficient flow permit migration to the upper river (Weitkamp and Ruggerone 2000).

Water withdrawal can reduce streamflow, inhibiting upstream migration (King County 1999c). Additionally, low dissolved oxygen concentrations may also inhibit upstream migration. Miller and Stauffer (1967) showed adult chinook in the Green River system avoid areas of low dissolved oxygen concentrations and high temperatures. However, Warner and Fritz (1995) reported that dissolved oxygen concentrations in the LDW were higher than in previous studies and attribute this

improvement to diversion of effluent (and its associated biological oxygen demand) from the Renton wastewater treatment plant in the 1980s.

In the mid-1970s, the Washington Department of Fish and Wildlife (WDFW) established an escapement goal of 5,800 naturally produced fall chinook using average escapement of natural and hatchery strays from 1965-1976 (Ames and Phinney 1977, as cited in Weitkamp and Ruggerone 2000). Based on spawning surveys, WDFW's estimated spawning escapement for naturally reared fish between 1968-1999 averaged 7,229 fish. The WDFW escapement goal was exceeded during 12 (40%) of 30 years. For the period from 1989-99, spawning escapements have been relatively high, averaging 8,578 fish, exceeding the WDFW goal for eight of the ten years (WDFW unpublished data, as cited in King County 1999c). Recent coded wire tag data suggest the percentage of the spawning ground population represented by hatchery strays may exceed 25% (Weitkamp and Ruggerone 2000). The contribution of Green River chinook salmon to the total chinook run entering Puget Sound and the Strait of Juan de Fuca ranged from 1.9 to 7.0% for the period 1979 to 1984 (Grette and Salo 1986).

Based on timing and the size of fish caught by beach seine, juvenile chinook salmon appear to use the LDW in two different life history trajectories: fingerlings that rear in the Green River and fry/fingerlings that rear in the LDW[13] (Warner and Fritz 1995; King County et al. 2000). Juvenile chinook can be found in the estuary from mid-February through early September, with a peak in abundance in late May, coinciding with hatchery releases (Warner and Fritz 1995). Beach seining data show a majority of outmigrant chinook enter the LDW in late May when they are 70-80 mm long coinciding with hatchery releases. Limited coded wire tag data suggest that fish of this size class are primarily hatchery fish along with some wild-spawned fish. Based on the duration of peak beach seine catches, individual juveniles of this size class likely move through the LDW in only a few weeks (Warner and Fritz 1995; Weitkamp and Schadt 1982).

Naturally spawned chinook of the fry/fingerling trajectory enter the estuary in late April and early May in small numbers[14] at approximately 45-60 mm in length. A continuous increase in size of beach-seined chinook during this period suggests these fish rear in the LDW for approximately 30 days, growing to 70-80 mm before migrating from the LDW (Warner and Fritz 1995). It should be noted that no tagging studies or mark-recapture studies have been conducted to conclusively establish the residence time of individual chinook salmon juveniles in the LDW. A study is currently being conducted to assess the ability of otolith sampling to provide insight to juvenile salmonid residence time in the LDW and other areas (Ruggerone 2002).

---

[13] An additional life history trajectory is fry that are washed out of the upper river into the estuary by high water events. Warner and Fritz (1995) caught few fish of this type. It is believed that this is an infrequent life history trajectory.

[14] Fewer than one fish per seine-set on average as compared with peak sampling of 100 fish per seine-set based on at least 20 sets per two- week period from February through September (Warner and Fritz 1995).

Juvenile chinook within the LDW tend to be associated with lower salinity and finer-grained sediment environments (Warner and Fritz 1995). Warner and Fritz (1995) showed that throughout the period of outmigration, relatively higher densities of juvenile chinook were caught in beach seines at upstream sites (Turning Basin 3 and above) that had lower salinity and finer grained sediments than at downstream sites. The size of juvenile chinook caught in any given season was no different at various beach seine sites throughout the LDW, suggesting fish are moving throughout the LDW during rearing (Warner and Fritz 1995).

Gut content analysis showed that in April/May, juvenile chinook prey predominantly on benthic species such as *Corophium* spp. (amphipods) and *Cumella vulgaris* and drift species such as adult dipterans (Cordell et al. 1997, 1999). Gut content analysis of fish caught in late May and June suggest juvenile chinook prey predominately on drift organisms such as wasps and ants (Cordell et al. 1997, 1999). Other prey, constituting over 25% of prey weight for any single site and date from April 1996, included collembolans, fish larvae, bivalve (clam) siphons, dipteran flies, polychaete and oligochaete annelid worms, and barnacle nauplius larvae (Cordell et al. 1997). These results are consistent with studies of chinook from other areas that show similar prey preferences (Meyer et al. 1981; MacDonald et al. 1987).

Coho salmon

Green River coho constituted from 0.9 to 1.4% of the total coho run entering Puget Sound and the Strait of Juan de Fuca for the period 1979-1984 (Grette and Salo 1986). Production was from hatcheries, naturally spawning hatchery-reared fish, and naturally spawning native fish (Grette and Salo 1986; WDFW 1993).

Adult coho return to the LDW between August and January, move through the LDW in a few days, and spawn and rear in all accessible reaches of the Green River drainage (Williams et al. 1975, as cited in Grette and Salo 1986). Juvenile coho rear in the Green River and move quickly through the LDW estuary as smolts (Weitkamp and Schadt 1982; Warner and Fritz 1995). The timing of outmigration is dependent on releases from Green River hatcheries (Weitkamp and Schadt 1982; Warner and Fritz 1995).

Chum salmon

The current status of the native chum population of the Green River watershed is unknown, but this population is suspected to have declined dramatically (Grette and Salo 1986; WDFW 1993). WDFW (1993) reported the state of the Green/Duwamish chum stock as unknown, whereas Nehlsen et al. (1991) reported the stock as at risk of extinction. Chum from the Green River constitute an insignificant portion of the total south Puget Sound run, and are not specifically addressed with harvest strategies for south Puget Sound stocks (Grette and Salo 1986).

Outmigrating chum salmon are reported to spend from several days to two months rearing in the Duwamish River estuary prior to moving offshore (Grette and Salo 1986). Warner and Fritz (1995) captured juvenile chum in beach seines throughout the

LDW from February through September 1994 and showed a continuous increase in size of fish captured, suggesting a relatively extended residence time in the LDW. Contradictory to this, in the same study, catch rates declined rapidly following peak catches, suggesting that fish were likely moving through the estuary within a few days. Adult chum salmon return to the LDW between September and December.

Gut content analysis showed that in the LDW, juvenile chum preyed on both epibenthic species and drift insects during outmigration, with a large temporal variation in prey composition (Cordell et al. 1997, 1999).

Pink salmon

Pink salmon appear infrequently and in low numbers. A run of odd-year pink salmon existed in the Green River in the 1930s (Williams et al. 1975, as cited in Grette and Salo 1986), though this run is believed to be currently extinct (Grette and Salo 1986). Warner and Fritz (1995) captured a total of 14 juvenile pink salmon in beach seines from nine stations throughout the LDW sampled approximately every two weeks from February through September 1994. Grette and Salo (1986) suggest that pink salmon have a high incidence of straying and that the few pink salmon captured in Green/Duwamish River are due to strays from other systems.

Sockeye salmon

There is limited evidence that sockeye salmon spawn and rear in the Green River watershed (Jeanes and Hilgert 2000). Juvenile sockeye appear to have the shortest residence time in the nearshore of all salmon species (Kerwin and Nelson 2000).

In summary, the LDW is used by a number of anadromous salmon species as a corridor for outmigration as juveniles and as a migration corridor for adults as they return to the upper watershed to spawn. Of the salmon species, chinook salmon have been studied the most extensively, and are listed as threatened under ESA.

A.2.2.3.2 Other salmonids

The Coastal-Puget Sound population of bull trout was proposed for listing under the federal ESA in June 1998 and was formally listed as threatened on November 1, 1999. The decline of bull trout has been primarily attributed to habitat degradation and fragmentation, blockage of migratory corridors, poor water quality, past fisheries management practices, and the introduction of non-native species (64FR 210: 58910-58933). Bull trout were historically found in the LDW, but current stock status is unknown (WDFW 2000). Because bull trout are morphologically similar to other char, positive identification of bull trout requires genetic testing. Thus, from a regulatory perspective, any char are assumed to be bull trout. Muckleshoot tribal biologists captured one char positively identified as an adult bull trout during beach seining in the LDW on May 24, 1994 during the period of peak juvenile salmon outmigration. However, it is unknown whether the fish reared in the Green River or was an opportunistic resident (Warner and Fritz 1995). Eight subadult bull trout ranging in length from 271 to 373 mm were captured in beach seines in Turning Basin 3 during

two sampling events in August and September 2000 (Shannon 2001). Peak numbers of juvenile shiner surfperch were captured at the same site the previous week, and near-peak numbers of shiner surfperch were captured in the same sampling in which the bull trout were caught (Shannon 2001). The co-occurrence of bull trout with high abundance of potential prey suggest that they may be opportunistically occupying the LDW to prey on these small fish. There is no evidence that bull trout are spawned or reared within the LDW. Bull trout juveniles typically remain in the upper tributaries for a period of two to three years prior to migrating to saltwater during spring. Adults typically return to their native streams in summer and fall (Grette and Salo 1986).

Bull trout exhibit both resident and migratory life-history strategies (Rieman and McIntyre 1993, as cited in 64FR 210: 58910-58933). Resident bull trout complete their entire life cycle in the tributary (or nearby) streams in which they spawn and rear. Migratory bull trout spawn in tributary streams where juvenile fish rear from one to four years before migrating to either a lake (adfluvial), river (fluvial), or in certain coastal areas, to salt water (anadromous) to mature (Fraley and Shepard 1989; Goetz 1989, as cited in 64FR 210: 58910-58933). Resident and migratory forms may be found together, and it is suspected bull trout pass resident or migratory behavior characteristics to their offspring (Rieman and McIntyre 1993, as cited in 64FR 210: 58910-58933).

Summer steelhead (*Oncorhynchus mykiss*) is a non-native stock sustained by wild spawning of hatchery-reared fish (WDFW 1993). The run size is unknown, but approximated at a few hundred fish (WDFW 1993). Winter steelhead consists of wild and hatchery fish with annual returns of 944-2,378 fish (WDFW 1993). Winter steelhead return to the Green River from December through May. Spawning generally begins about mid-March and continues to early June, with a peak in mid-May (Cropp 1985, as cited in Grette and Salo 1986). Grette and Salo (1986) report that repeat spawners make up approximately 19% or less of returning wild adults in the Green River (1976/77 to 1983/84). Summer steelhead outmigrate from the Green River after rearing for two years as smolts, and do not have an extensive residence time in the LDW. Winter steelhead outmigrate from the Green River as subyearling adults and also do not rear extensively in the LDW.

Sea-run cutthroat trout may exist in the LDW, but little is known about this population. A total of 11 adult cutthroat trout were captured in beach seines at nine stations sampled approximately 30 times each throughout the LDW from February through June 1994 (Warner and Fritz 1995). In Washington, adult cutthroat return to their home stream from July to January, with the peak occurring in October and November (Wydoski and Whitney 1979). Smolt outmigration occurs from April through May (Wydoski and Whitney 1979).

In summary, winter steelhead appear to be the most numerous species among piscivorous salmonids in the LDW. Bull trout and cutthroat trout also use the LDW, but relatively little is known about their populations.

A.2.2.3.3 Non-salmonid fishes

The most abundant non-salmonid fish in the LDW are snake prickleback, shiner surfperch, English sole, Pacific staghorn sculpin, starry flounder, and longfin smelt (Matsuda et al. 1968; Miller et al. 1975; Miller et al. 1977a; Weitcamp and Campbell 1980; Meyer et al. 1981; Warner and Fritz 1995; Taylor et al. 1999; West et al. 2001; Robertson 2002). English sole and shiner surfperch are reported to be most abundant in the lower portion of the estuary, and starry flounder most abundant in the upper estuary according to trawl data (Miller et al. 1975; Matsuda et al. 1968). Longfin smelt, snake prickleback, and Pacific herring are seasonally abundant in the LDW. Snake prickleback were essentially absent from beach seine samples conducted in the lower section of the LDW from September through the end of April but were abundant mid-May through August (Weitcamp and Campbell 1980). Miller et al. (1977a) note that in otter trawls, snake prickleback were abundant all year, but were particularly numerous during summer months. During this period of high abundance, snake prickleback were rare below Kellogg Island but were abundant at six stations between Kellogg Island and Turning Basin 3 (Miller et al. 1977a). Otter trawl data show peaks in longfin smelt abundance in fall, early winter, and summer (Miller et al. 1977a). Miller et al. (1977a) suggest that the fall-winter peak (80-115 mm fish) may represent part of a spawning run and that the late summer peak (30-50 mm fish) represents downstream migrant young of the year. Pacific herring were reported to be present in purse seine samples throughout the year but were present in beach seine samples only in November and December (Weitkamp and Campbell 1980). Pacific herring captured using both gear types were reported to be small fish (Weitkamp and Campbell 1980). Pacific staghorn sculpin and starry flounder are year-round residents. Fish in the LDW exhibited similar relative abundances among the ten studies noted above.

In all studies, Pacific staghorn sculpin was consistently one of the most abundant fish captured in the LDW (Table A-2-4). Miller et al. (1977a) reported that Pacific staghorn sculpin were abundant in otter trawls all seasons but were particularly abundant in fall. However, in the summer, Pacific staghorn sculpin were absent below approximately 3.2 km upstream (RM 2) (Miller et al. 1977a). Weitcamp and Campbell (1980) reported that Pacific staghorn sculpin taken in the fall and winter in beach seines and otter trawls were primarily small adults or juveniles of 50-150 mm. In the spring, Pacific staghorn sculpin were more abundant as small fish of 11-30 mm. Few Pacific staghorn sculpin larger than 150 mm were collected in beach seines by Weitcamp and Campbell (1980). Approximately two to ten Pacific staghorn sculpin per two-hour set were collected in floating gill nets at the south end of Kellogg Island (Weitcamp and Campbell 1980). Because of the selectivity of this gear type, these fish were probably large enough to be capable of eating small fish. Pacific staghorn sculpin are opportunistic feeders. They feed mostly on crabs, shrimps and amphipods, but also take larval, juvenile and adult fishes, as well as polychaete worms, mollusks and other invertebrates (Fitch and Lavenberg 1975). Larger sculpin are more likely to eat at a higher trophic level.

Shiner surfperch abundance peaks in summer during the bearing of young (Miller et al. 1975). Shiner surfperch are opportunistic omnivores, feeding on zooplankton, small crustaceans, algae, and detritus (Gordon 1965; Bane and Robinson 1970), and also on polychaetes, mollusks, and benthic organisms (Boothe 1967; Barry et al. 1996).

English sole are common in the LDW over all seasons, with peak abundance in spring (Miller et al. 1977a). In Puget Sound, English sole are typically found on soft sand or mud bottoms at depths of 25 to 50 m (Smith 1936). In this habitat, juvenile English sole (those less than 110 mm) eat annelids (Smith 1936), copepods, amphipods, and mollusks (Holland 1954). Adult English sole studied in British Columbia were found to eat clams, clam siphons, small mollusks, marine worms, small crabs, small shrimps, and brittle stars. It has been suggested that English sole exist in discrete populations with some site fidelity. Day (1976) conducted a tagging study that showed that some fish released dozens of miles from their capture site in Puget Sound returned to the original capture area within one year, although the results were based on a low recapture rate. Fish not displaced during tagging remained essentially at the original area of capture. In central Puget Sound, adult populations of English sole concentrate in Elliott Bay and Port Gardner to spawn, but disperse after spawning, which usually occurs in winter (Pallson 2001). English sole migrate seasonally to their spawning grounds in Puget Sound in winter (Forrester 1969) and typically spawn in Puget Sound during February and March (Smith 1936). Angell et al. (1975) reported off-season migration in winter and spring of all age groups of central Puget Sound fish from Meadow Point to Carkeek Park (northwest side of Seattle) at depths of 3 to 30 m. Juveniles (10 to 25 mm standard length), not all completely metamorphosed, migrated from spawning areas to nursery grounds to begin settling in December or May and June (King County 1999c). Data from Malins et al. (1982) show that during the winter and spring, greater than 50% of the English sole in the LDW are juveniles (<150 mm).

In summary, the LDW provides habitat for anadromous salmonids and resident fish. Anadromous salmon are present during rearing and migration to and from spawning sites in the upper watershed. In the LDW, juveniles make up an important part of the food web, preying on various epibenthic, water column, and drift organisms, and serving as prey for larger fish and wildlife. Of the non-salmonid fishes, shiner surfperch, English sole, Pacific staghorn sculpin, snake prickleback, longfin smelt, and starry flounder are among the most abundant species in the LDW. Seasonal abundance of fish in the LDW varies, peaking in the summer and early fall. Fish in the LDW are primarily carnivorous and appear to rely extensively on the epibenthic invertebrates.

A.2.2.4 Wildlife

The aquatic and semi-aquatic habitats of the LDW support a diversity of wildlife species. Formal studies, field observations, and anecdotal reports indicate that up to 87 species of birds and 6 species of mammals utilize the LDW during at least part of

the year to feed, rest, or reproduce. This section provides an overview of these bird and mammal species.

A.2.2.4.1 Birds

The bird species associated with the LDW are presented in Table A-2-5. The birds using the site can be grouped as follows:

- passerine/upland birds
- raptors
- shorebirds/waders
- waterfowl
- seabirds

Canning et al. (1979) conducted extensive surveys of the avifauna of Kellogg Island, as well as occasional surveys of the entire LDW from September 1977 to July 1978. They recorded a total of 70 species: 26 passerines/upland birds, 3 raptors, 11 shorebirds/waders, 17 waterfowl, and 13 seabirds. They report Kellogg Island had a much higher diversity of birds than the rest of the LDW due to its seclusion and greater variety of habitats.

Cordell et al. (1999) monitored bird populations monthly from 1995 to 1997 at four sites: two sites in Turning Basin 3, one on Kellogg Island, and one at Terminal 105. They recorded 75 species of birds: 32 passerine/upland birds, 7 raptors, 8 shorebirds/waders, 16 waterfowl, and 12 seabirds. Diversity and abundance were highest at the Kellogg Island site, but other areas of the LDW were also consistently used by a wide variety of birds. Birds were most abundant in the spring and least abundant in the summer. The following provides a brief summary of site usage by the various types of bird species in the LDW.

Table A-2-5. Bird species using the LDW

Common Name	Latin Name	Common Name	Latin Name
Passerine/Upland species		**Raptors**	
Blackbird, red-winged	*Agelaius phoeniceus*	Eagle, bald	*Haliaeetus leucocephalus*
Bushtit, common	*Psaltriparus minimus*	Falcon, peregrine	*Falco peregrinus*
Chickadee, black-capped	*Poecile atricapillus*	Hawk, Cooper's	*Accipter cooperii*
Cowbird, brown-headed	*Molothrus ater*	Hawk, red-tailed	*Buteo jamaicensis*
Crow, northwestern	*Corvus corinus*	Hawk, sharp-shinned	*Accipiter striatus*
Dove, rock	*Columba livia*	Hawk, Swainson's	*Buteo swainsoni*
Finch, house	*Carpodacus mexicanus*	Merlin	*Falco columbarius*
Flicker, northern	*Colaptes auratus*	Osprey	*Pandion haliaetus*
Goldfinch, American	*Spinus tristis*	**Waterfowl**	
Hummingbird, Anna's	*Calypte anna*	Bufflehead	*Bucephala albeola*
Junco, dark-eyed	*Junco hyemalis.*	Canvasback	*Aythya valisineria*
Kingfisher, belted	*Ceryle alcyon*	Coot, American	*Fulica americana*
Kinglet, ruby-crowned	*Regulus calendula*	Duck, domestic	*Anas domesticus*
Siskin, pine	*Spinus pinus*	Gadwall	*Anas strepera*
Quail, California	*Lophortyx californicus*	Goldeneye, Barrow's	*Bucephala islandica*
Robin, American	*Turdus migratorius*	Goldeneye, common	*Bucephala clangula*
Sparrow, English (house)	*Passer domesticus*	Goose, cackling Canada	*Branta canadensis minima*
Sparrow, fox	*Passerella iliaca*	Goose, Aleutian	*Branta canadensis*
Sparrow, golden-crowned	*Zonotrichia atricapilla*	Goose, domestic	*Branta domesticus*
Sparrow, savannah	*Passerculus sandwichensis*	Mallard	*Anas platyrhynchos*
Sparrow, song	*Melospiza melodia*	Merganser, common	*Mergus merganser*
Sparrow, white-crowned	*Zonotrichia leucophrys*	Merganser, hooded	*Lophodytes cucullatus*
Starling, European	*Sturnus vulgaris*	Merganser, red-breasted	*Mergus serrator*
Swallow, barn	*Hirundo rustica*	Scoter, surf	*Melanitta perspicillata*
Swallow, cliff	*Petrochelidon pyrronota*	Teal, greenwinged	*Anas carolinensis*
Swallow, tree	*Iridoprocne bicolor*	Wigeon, American	*Mareca americana*
Swallow, violet-green	*Tachycineta thalassina*	**Seabirds**	
Thrush, Swainson's	*Hylocichla ustulata*	Cormorant, double-crested	*Phalacrocorax auritus*
Towhee, rufous-sided	*Pipilo erythrophthlamus*	Cormorant, pelagic	*Phalacrocorax pelagicus*
Warbler, orange-crowned	*Vermivora celata*	Grebe, eared	*Podiceps capsicus*
Wren, Bewick's	*Thryomanes bewickii*	Grebe, horned	*Podiceps auritus*
Wren, house	*Troglodytes aedon*	Grebe, pied-billed	*Podilymbus podiceps*
Shorebirds/Waders		Grebe, red-necked	*Podiceps grisegena*
Dowitcher	*Limnodromus sp.*	Grebe, western	*Aechmophorus occidentalis*
Dunlin	*Erolia alpina*	Guillemot, pigeon	*Cepphus columba*
Heron, great blue	*Ardea herodias*	Gull, glaucous-winged	*Larus glaucescens*
Heron, green	*Butorides virescens*	Gull, mew	*Larus canus*
Killdeer	*Charadrius vociferus*	Gull, ring-billed	*Larus delawarensis*
Sanderling	*Crocethia alba*	Loon, common	Gavia immer
Sandpiper, least	*Calidris minutilla*	Loon, Pacific	Gavia Pacifica
Sandpiper, spotted	*Actitis macularia*	Loon, red-throated	Gavia stellata
Sandpiper, western	*Calidris mauri*	Murre, common	*Uria aalge*
Yellowlegs, lesser	*Totanus flavipes*	Tern, Caspian	*Hydroprogne caspia*

A.2.2.4.2 Passerines/upland birds

Thirty-two species of passerine/upland birds have been documented along the LDW (Canning et al. 1979; Cordell et al. 1999). These birds, while generally associated with upland habitats, occasionally forage in the exposed mudflats or use freshwater habitats along the river for bathing (Canning et al. 1979). Because they primarily use upland habitat, passerine birds likely experience very limited exposure to contaminated sediments in the LDW.

A.2.2.4.3 Raptors

Eight raptor species have been reported to use the LDW (Cordell et al. 1999). The bald eagle is listed under ESA as a threatened species, but is currently under review for delisting. In Washington, it is also listed as a state threatened species (WDFW 2001). There are five nests within five miles of the LDW that were occupied in 1999 (King County 1999d). The closest nest is located in West Seattle within 1.6 km (1 mi) of the LDW. One or two pairs of resident eagles may be found in the LDW vicinity during the summer (King County 1999c). Overwintering migrant eagles are routinely observed in the vicinity of the LDW from the beginning of October through late March.

The bald eagle is an opportunistic forager with site-specific food habits based on available prey species (Buehler 2000). Bald eagles consume dead and live fish, birds, and mammals extensively. In most regions, bald eagles seek out aquatic habitats for foraging and prefer fish (Buehler 2000). Spawned-out salmon are a particularly important food item for eagles in the Pacific Northwest, though not in the LDW because returning salmon spawn farther upstream. Of 45 fish identified in a study of prey remains at the base of eagle nest trees throughout Puget Sound, eight were rockfish, 10 were starry flounder, and the remainder included cod, pollock, hake, cabezon, red Irish lord, sculpins, surfperch, salmon, plainfin midshipman, and channel catfish (Knight et al. 1990). Although eagles feed primarily on fish, birds, such as grebes, gulls, and waterfowl, make up a portion of their diet during winter months. Eagles have been reported to kill western grebe in the Duwamish River during winter (Strand 1999, as cited in King County 1999b). Eagles also have been reported to prey on great blue heron chicks (Norman et al. 1989, as cited in King County 1999b).

Cooper's and sharp-shinned hawks have been observed to overwinter in the LDW. These relatively small raptors generally feed on birds up to the size of quail. They may rarely feed on aquatic birds (Canning et al. 1979; Cordell et al. 1999). Red-tailed hawks, a resident species commonly observed along grassland/woodland margins along the LDW, feed primarily on rodents but have been noted to pursue ducklings in the study area. Swainson's hawks and merlin are rare in the LDW and not likely to prey on aquatic associated species (Canning et al. 1979; Cordell et al. 1999).

Cordell et al. (1994) report osprey using Kellogg Island and the restored turning basin sites. An osprey nest is located on a utility pole near Terminal 105 (Matt Luxon personal observation 6/00). Ospreys feed opportunistically and almost exclusively on

live fish from fresh or salt water. Ospreys can penetrate only about 1 m below the water surface. Therefore, they generally catch only surface fish or those that frequent shallow flats and shorelines.

Reportedly, a female peregrine falcon recently attempted but failed to nest at the West Seattle bridge and to mate with the male falcon inhabiting the Washington Mutual Tower in downtown Seattle (Anderson 2002). Peregrine falcons prey primarily on songbirds, shorebirds, waterfowl, and seabirds. The peregrine falcon is listed as a species of concern under ESA. WDFW currently lists peregrine falcon as a state endangered species, although they are recommending changing the listing to a state sensitive species due to increased breeding success.

A.2.2.4.4 Shorebirds/waders

Eight species of shorebirds and wading birds have been documented in the LDW (Cordell et al. 1999), including green heron and great blue heron. Of these species, great blue heron make up the only sizeable or consistent population.

The great blue heron is a semi-aquatic wading bird that has a range from the coasts of southeast Alaska and Northern British Columbia, through Canada and the US, and south to Belize and Guatemala. The great blue heron is found primarily in natural wetlands and along riverbanks, but can also be found in brackish marshes, lagoons, lakes, and along ocean shores. They were the most abundant shore/wading bird recorded by Cordell et al. (1996) on the LDW, and are a year-round resident. Great blue heron nest in colonies of up to several hundred pairs, preferably on islands or wooded swamps (Butler 1992). A heron colony of up to 37 active nests was located in West Seattle a few hundred meters from Kellogg Island until 1999, but no successful nesting occurred there in 2000 or 2001 (Norman 2002). Other heron colonies in the vicinity of the LDW are located about 12 km (7.5 mi) south in Renton and 11 km (6.8 mi) northwest near Salmon Bay.

Great blue heron feed in shallow water primarily on small fish, such as juvenile salmonids, but they also take crustaceans, insects, amphibians, reptiles, and occasionally small mammals (Kushlan 1978; Butler 1992). Great blue heron hunt by sight and stalk or ambush their prey. They will also feed by probing, quickly moving their bills in and out of the water and substrate. Great blue heron feed on small fish that range in size from 8–33 cm (Kirkpatrick 1940; Alexander 1977; Hoffman 1978). Butler (1992) reports that shiner surfperch is a major food source for female and hatchling great blue heron and may be important for juvenile survival.

The two most common shorebirds observed in the LDW are sandpipers and killdeer. The spotted, least, and western sandpipers are reported to use the LDW in substantial numbers. These birds feed on insects, small crustaceans and mollusks, worms, and other invertebrates, and rarely on seeds and berries. Spotted sandpiper feed occasionally on small fish and carrion (Oring et al. 1983). Sandpipers are presumed to feed in the intertidal mudflats along the LDW. Least and western sandpipers occur in

mixed flocks and are difficult to distinguish. These species nest primarily in northern Canada and Alaska in the summer months (Paulson 1993), but are reported to frequent Kellogg Island from September through May (Canning et al. 1979). Most are thought to be migrants, though some may reside in the LDW throughout the winter.

Spotted sandpipers are a common bird in western Washington, and are known to nest along the LDW. They have been observed in the LDW from late June through September (Cordell et al. 1996) but have been known to overwinter locally (Paulson 1993). Nesting birds arrive in May and June. Canning et al. (1979) recorded seven spotted sandpiper nests located on Kellogg Island and at least three additional nest sites were suspected. Spotted sandpipers breed in open habitats along the margins of water bodies (Oring and Lank 1986).

Killdeer, another common shorebird, feed in intertidal mudflats. Their diet includes small invertebrates, insects, and some vegetative matter. Killdeer are a common bird that uses the LDW year-round, with 20 to 60 birds reported to use the area in the winter and approximately 10 in the area in the fall and spring. They are reported to nest along the LDW, though few are reported outside the Kellogg Island area (Canning et al. 1979).

A.2.2.4.5 Waterfowl

Cordell et al. (1999) reported 16 species of waterfowl utilizing the LDW, including nine species of dabbling ducks. All species are migratory, though some non-migratory populations exist. In general, these birds overwinter in the Puget Sound area (and farther south) and migrate north in the summer. The dabbling ducks feed on aquatic plants, seeds, and grasses and to some extent small aquatic animals and insects. Feeding occurs primarily in shallow water and over intertidal mudflats. A resident population of approximately 25 mallards lives year-round in the LDW, and an additional population of approximately 15 mallards overwinters in the LDW. As many as 290 migratory mallards have been reported to move through the LDW (Canning et al. 1979). The other dabbling duck species use the LDW for nesting and migration. The most significant of these are gadwalls. Approximately ten gadwall nests have been observed along the LDW in the vicinity of Kellogg Island (Canning et al. 1979).

Canvasback, greater scaup, bufflehead, and common and Barrow's goldeneye are reported to use the LDW. These birds dive for small aquatic animals and plants. Canvasback feed primarily on plants, scaup on equal portions of plants and animals, and bufflehead and goldeneyes exclusively on aquatic animals and insects. A peak population of approximately 60 canvasbacks arrives in the LDW in November and departs in late February, using Kellogg Island as a primary feeding area. Greater scaup and common and Barrow's goldeneyes arrive in the study area in late November and depart by early May. A small population of approximately eight buffleheads is reported to overwinter in the LDW from December to May. Feeding by all diving duck species is centered around Kellogg Island (Canning et al. 1979).

All three species of North American mergansers have been recorded to use the LDW, two substantively. Migratory common mergansers are reported to use the LDW from September to March, though none overwinter in the area. Approximately 30 red-breasted mergansers are reported to overwinter in the LDW from December to March. These birds feed primarily on small fish and are reported to feed in the deeper water of the channel (Canning et al. 1979).

A resident population of approximately 1,000 Canada geese resides in the vicinity of Lake Washington. The Duwamish population is thought to be a part of the Lake Washington population. Migratory Canada geese arrive in the LDW in January and February and remain until the end of July as a spring nesting population. Canada geese swim in the LDW and feed in intertidal habitats. They feed primarily on grass and terrestrial vegetation (Canning et al. 1979). In the LDW, 40 to 50 birds overwinter from September to April along Kellogg Island and the west bank of the waterway along the South Park district and in Turning Basin 3 (Canning et al. 1979).

A.2.2.4.6 Seabirds

Thirteen species of seabird have been recorded in the LDW (Canning et al. 1979; Cordell et al. 1999), including two species of cormorants (pelagic and double-crested). Cormorants feed primarily on small fish and occasionally crustaceans. Wintering cormorants use the LDW from November to May, with large numbers present from December to April (Canning et al. 1979; Cordell et al. 1996).

Several species of gulls are reported to use the LDW. Gulls feed on fish and shellfish and are omnivorous scavengers. Glaucous-winged gulls and mew gulls are the only species reported to use the area in large numbers. Glaucous-winged gulls are reported to use the area throughout the year. Mew gulls frequent the area, occasionally in large numbers, from September through May (Canning et al. 1979).

Caspian terns have been seen using Kellogg Island (M. Luxon personal observation). Pigeon guillemots and common murres have been reported in the LDW; however, their use of the LDW is infrequent. These birds feed primarily on pelagic fish, though bottomfish and crustaceans may also be taken.

Common loons are a state candidate species under review for listing as threatened or endangered (WDFW 2001). They are present in Puget Sound in winter and use local waters for resting during migrations to and from wintering areas farther south. Their diet consists primarily of small fish and other aquatic animals. Annual winter counts indicate 10 to 30 birds in the Seattle area, although they are reported to be rare visitors to the LDW (Canning et al. 1979).

Three species of grebe are reported in the LDW. Of these, only western grebes are found in substantial numbers. Grebes and other marine bird species have been declining in recent years (Nysewander et al. 2001). Feeding behavior varies with species. In marine waters, the eared grebe primarily takes crustaceans while the western grebe favors fish. The most common fish species taken by western grebes are

Pacific herring, pilchard, stickleback, sculpin, sea perch, and smelt. Western grebes occasionally feed on juvenile salmonids. The LDW population was estimated to comprise about 90 birds in the 1970s (Canning et al. 1979). Grebes arrive in the LDW from October to November and depart by early May.

In summary, the LDW is a corridor frequented by a diverse avian group. It is utilized mostly by shore birds, waders, seabirds, and waterfowl, which feed in areas of mudflat and other shallow-water habitat. Raptors also use the LDW for foraging.

A.2.2.4.7 Mammals

Three marine mammal species may occasionally enter the LDW: harbor seal, California sea lion, and harbor porpoise (Dexter et al. 1981). Harbor seals and California sea lions have been recently observed in the LDW (WDFW 1999), but recent information on harbor porpoise usage was not available. The harbor seal can be found along both North American coasts (Hoover 1988; Payne and Selzer 1989). Along the Pacific coast they can be found from Alaska to Baja California and mainland Mexico, and are the most commonly observed pinniped species (Hoover 1988). Along the Pacific coast, they can be seen in protected harbors year-round (Boulva and McLaren 1979). Harbor seals are commonly seen in Elliott Bay and occasionally enter the LDW (Kenney 1982).

Harbor seals are opportunistic feeders, selecting prey based on availability and ease of capture (Pitcher and Calkins 1979; Pitcher 1980; Schaffer 1989). Their diet can vary seasonally and includes bottom dwelling fishes, invertebrates, and species that congregate for spawning (Pitcher and Calkins 1979; Everitt et al. 1981; Lowry and Frost 1981; Roffe and Mate 1984). In Washington, the most important prey include Pacific whiting, tomcod, walleye pollock, flatfishes, Pacific herring, shiner surfperch, plainfin midshipman, and sculpins (NMFS 1997). Fish consumed are generally between 40 and 280 mm (Brown and Mate 1983). Harbor seals have been shown to forage over large areas ranging from 5 km (Stewart et al. 1989) to 55 km (Beach et al. 1985).

California sea lions and harbor porpoises are also opportunistic feeders, consuming various fish species depending on availability (Marine Mammal Center 2000). California sea lions and harbor porpoises will, like harbor seals, also feed on non-fish species such as squid and octopus (Yates 1998).

A survey of sea lions and harbor seals was conducted in the LDW from December 1998 to June 1999 (WDFW 1999). This survey monitored the presence of sea lions and harbor seals in the East and West Waterways and in the LDW up to the 16th Avenue South bridge for a total of 307 hours on 52 days. In the LDW, sea lions were observed on 16 occasions and seals on 17 occasions, with most observations for both species occurring below the 1st Avenue South bridge. In the East and West Waterways, sea lions were observed 69 times and seals 6 times; both species used the West Waterway most frequently.

Three species of semi-aquatic terrestrial mammals use the LDW: raccoons, muskrats, and river otters. Raccoons are reported to be common along the forested ridge slopes to the west of the LDW. Raccoons are scavengers that feed on carrion and occasionally on fish. Muskrat populations are reported to exist at Terminal 107 and at Turning Basin 3 (Canning et al. 1979). Muskrats are herbivores, feeding primarily on aquatic and semi-aquatic plants.

Anecdotal information indicates that a river otter family lives year-round on Kellogg Island in the LDW, although otters were not observed by Cordell during wildlife surveys (Cordell 2001b). River otters are almost exclusively aquatic and prefer food-rich habitats such as the lower portions of streams and rivers, estuaries, and lakes and tributaries that feed rivers (Tabor and Wight 1977; Mowbray et al. 1979). Local river otters feed primarily on fish but will also feed on crabs and sometimes mussels and clams (Strand 1999, as cited in King County 1999b). River otters range over an area sufficiently large enough for foraging and reproduction (Melquist and Dronkert 1987); however, they are typically found in a limited number of activity centers within their overall range. In streams, the river otter's home range can average 30 km (Melquist and Hornnocker 1983).

In summary, the LDW corridor provides habitat for a limited number of mammal species. It may serve as a significant part of the home range of a river otter family, but is used only occasionally as a foraging site by marine mammals.

A.2.2.5 Plants

Three types of plants play key roles in maintaining high productivity in estuaries: 1) phytoplankton suspended within the photic zone of the water column; 2) benthic microflora (microscopic plants) living on the sediment surface wherever sufficient light reaches the bottom; and 3) macroflora (rooted plants) and periphyton growing in shallow water and along the shoreline. These plants are the foundation of the complex food webs found in estuaries such as the LDW. The macroflora and periphyton provide nursery habitat for fish and shellfish. High estuarine productivity results in food chains that can be quite long, extending to six or seven trophic levels.

Few surveys have investigated the plant communities present in the LDW (Cordell et al. 2001; USFWS 2000; Tanner 1991; Canning et al. 1979). The methods used to assess plant communities ranged from analysis of aerial photos to field surveys. Many of these surveys were conducted to investigate habitat availability in the LDW and mainly addressed the plant communities of tidal marsh areas. Thus, this section will focus on macrophyte species of tidal marsh areas in the LDW.

Tidal marsh plant assemblages are tolerant of a narrow range of salinity. Thus, tidal elevation and salinity gradients determine the potential distribution for estuarine plants. Intertidal elevation gradients between mean lower low water (MLLW) and mean higher high water (MHHW) create habitats such as low-, mid-, and high-elevation tidal marshes. Salinity gradients range from saline to brackish to fresh tidal

waters. The most productive areas for estuarine plant communities are found in tidal marshes. Marsh soils are generally fine-textured and nutrient-rich, and support grasses, sedges, rushes, and various other types of plants associated with marine and estuarine habitats. In the LDW, there is a total of 1.75 ha of habitat for macrophytes, primarily limited to portions of Kellogg Island and other small areas with vegetated intertidal habitat (USFWS 2000).

Carex and *Scirpus* are the predominant vegetation type between Turning Basin 3 and Kellogg Island. Downstream from Kellogg Island are more marine plants such as *Salicornia, Distichlis,* and *Atriplex*. The interior high marsh plant community of Kellogg Island, which is flooded only by higher spring tides, includes *Carex lyngbyei, Distichlis spicata, Juncus balticus* (Baltic rush), and *Phragmites* sp., a non-native species (Battelle et al. 2001). The naturally occurring *Carex* patches surveyed in 1993 occurred between elevations of 1.6 to 3.0 m (5.2 to 9.7 ft), and the single patch of naturally occurring *Scirpus* was at 3.7 m (12 ft) (Cordell 2001a). Thus, these plants are seldom present under water.

In summary, the plant community in the LDW corridor exists as remnant patches of tidal marsh dominated by *Carex* and *Scirpus* species. These patches provide habitat for the fish, avian, and wildlife species that utilize this area.

A.2.3 Receptor of Concern Selection

In this section, ROCs are selected to represent benthic invertebrates, fish, wildlife, and plant species based on a set of ROC selection criteria. Inherent to the ROC process is the realization that not all species in the LDW can be evaluated individually due to the large number and variety of species present. Instead, representative species are chosen to include species that are most exposed to contaminated sediment (sensitive species are also preferred, but the relative sensitivity of most species is not known). In this way, species not selected should also be protected.

A systematic process was followed to select representative species as ROCs based on the available information for the resources presented in Section A.2.2. This process is consistent with available EPA guidance and the process commonly used in Superfund risk assessments.

Key considerations in the selection of ROCs included:

- Potential for exposure to sediment-associated chemicals
- Human and ecological significance
- Site usage
- Sensitivity to COPCs at the site
- Data availability

To ensure that ROCs were selected to represent all potential exposure pathways for sediment-associated COPCs, key direct and indirect exposure routes from sediment

were identified (e.g., direct exposure to sediment or consumption of prey associated with sediment either directly or through its prey). Groups of organisms that may be exposed via these pathways were then identified. For example, benthivorous fish may be exposed through direct sediment contact *and* through the food chain, whereas pelagic piscivorous fish would primarily be exposed through the food chain only. Thus, representative species were selected from these groups of organisms believed to be most exposed. Next, human or ecological significance was considered (i.e., species valued by society, have special regulatory status [i.e., threatened or endangered], or serve a unique ecological function).

Site usage, sensitivity to COPCs at the site, and data availability were also evaluated to determine the final list of ROCs. Site usage is an important criterion because it determines the exposure of a species; species that occupy the LDW during a significant part of the year or during sensitive periods, such as nesting, were preferred. Sensitivity to COPCs was evaluated based on available toxicological data, although in many cases the availability of data specific to LDW resident species is low. Therefore, where necessary, toxicological information from surrogate species, or a wide range of species, was used because species-specific data are not available. Finally, data availability regarding both site-specific exposure and effects was assessed, and species for which there are related site-specific data (such as COPC concentrations in food, site usage, and feeding) and toxicological data (such as sediment toxicity tests) were preferred. The following sections provide additional rationale for each of the ROCs selected; a summary of the selected ROCs is provided in Section A.2.3.5.

A.2.3.1 Benthic invertebrates

As discussed in Section A.2.2.2, a wide variety of benthic invertebrates inhabit the LDW; most of these species are in direct contact with sediment year round and have a limited home range. Benthic invertebrates are an important food source for other invertebrates, fish, birds, and mammals, and provide essential nutrient cycling into the LDW. Thus, the diversity and abundance of benthic invertebrates is an important component of the ecosystem. In addition, benthic organisms have been shown to be susceptible to sediment-associated chemicals, and data are available to assess their exposure and predict or measure potential effects.

Benthic invertebrates use various techniques to nourish themselves, and thus may be exposed to sediment through somewhat different pathways (e.g., filter feeder vs. detritus feeder). Benthic organisms include sediment dwellers (benthic infauna) and organisms closely associated with the sediment surface (epibenthos).

Numerical chemical sediment quality standards (SQS) and cleanup screening levels (CSLs) promulgated in the Washington State Sediment Management Standards (SMS) are based on measured benthic invertebrate infaunal abundance and on the results of toxicity tests conducted with Microtox, oyster larvae, and amphipods. Recent surveys of the LDW (Striplin 1998; Cordell et al. 1999) demonstrate that many organisms present or expected to be present in the LDW are generally similar to those included in

the SMS toxicity test suite, although the suite of organisms in the SMS does not include representatives from all taxa of benthic invertebrates in the LDW. Because the SMS standards are based on the lowest apparent effects thresholds (AETs) (see Section A.3.2.1) for the various species and endpoints represented in the SMS suite, including a benthic community metric, they are designed to be protective of the benthic community as a whole. Thus the benthic community as a whole will be evaluated in this Phase 1 ERA as an ROC.

However, SMS were not developed to explicitly address issues associated with bioaccumulation of COPCs by benthic invertebrates. Although this issue is implicitly addressed through the incorporation of benthic community structure into the overall development of the SMS, the SQS and CSL do not specifically address risks to higher-trophic-level benthic invertebrate species, such as crabs. Therefore, crabs were also selected as an ROC to better represent the spectrum of benthic invertebrate species present in the LDW. Crabs have a relatively larger home range than most of the benthic invertebrate species covered by the SMS, and they are in a higher trophic position in the food web. In addition, sufficient (but limited) toxicological data for crab are available for a comparison with the limited chemical dataset available for crab tissue.[15] Using a tissue-based approach integrates all potential exposure pathways for crab.

Other than crab, for reasons discussed above, no specific benthic invertebrate species were selected as ROCs in this Phase 1 ERA. The availability of relevant toxicity data and the feasibility of collecting additional benthic invertebrate tissue data will be evaluated as part of the data gaps process to determine whether specific benthic invertebrate species (e.g., mussels) should be evaluated as part of the Phase 2 ERA.

A.2.3.2 Fish

The potential fish receptors of concern were grouped into the following three broad categories based on potential sediment exposure at the site:

- **Piscivorous fish**—including species such as sand sole, Pacific staghorn sculpin, and bull trout
- **Benthivorous fish**—including species such as English sole, rock sole, and starry flounder
- **Anadromous juvenile salmonids**—including juvenile chinook salmon and juvenile chum salmon

Omnivorous fish, such as shiner surfperch, are the only other broad category of fish receptors that are assumed to be less exposed to COPCs from the LDW through their diet and sediment ingestion than piscivorous or benthivorous fish. Primarily omnivorous fish typically consume algae and benthic invertebrates (e.g., zooplankton, small crustaceans and mollusks) (Miller et al. 1977b), and may incidentally ingest

[15] Limited resident and caged mussel tissue data were also available.

sediment. This exposure is assumed to be less than that of benthivorous fish, such as English sole, which may ingest significant amounts of sediment while foraging for benthic invertebrates. As stated above, omnivorous fish also ingest algae, an additional potential exposure pathway. The significance of this pathway depends upon the extent to which sediment-associated chemicals migrate through the water column, are taken up by algae, and then consumed in significant quantities. This pathway is assumed to be insignificant compared to other more direct pathways examined (e.g., incidental ingestion of sediment or contaminated benthic species). Using the criteria discussed in Section A.2.3, the following fish species were selected as ROCs in the LDW:

- wild juvenile chinook salmon – juvenile salmonids
- bull trout – piscivorous fish
- English sole – benthivorous fish

The remainder of this section discusses the rationale for selecting each ROC and how these species serve as surrogates for protection of other similar and important species within the LDW (Table A-2-4).

A.2.3.2.1 Wild juvenile chinook salmon

Wild juvenile chinook salmon (*Oncorhynchus tshawytscha*) were selected primarily because the Puget Sound evolutionary significant unit of chinook salmon (to which the Green River belongs) is a federally threatened species under ESA. In addition, they serve as a surrogate for other juvenile anadromous salmon. Juvenile chinook salmon serve as an appropriate representative for other salmonids because they are believed to have similar or greater estuarine residence times as other juvenile salmonids in the LDW. During their spring outmigration, juvenile chinook salmon are among the most abundant fish in the LDW and are an important prey item for birds and piscivorous fish in the LDW (Warner and Fritz 1995). Juvenile chum salmon are also present in large numbers in the LDW from April through June and may rear extensively in the LDW (Warner and Fritz 1995). Residence times of all species of juvenile salmonids in the LDW are uncertain; however, juvenile chinook salmon are generally regarded as the most estuarine-dependent juvenile salmonid and their exposure to sediment-associated chemicals is likely equal to or greater than that of other juvenile salmonids. Because chinook salmon spend only a brief part of their juvenile period in the LDW, their exposure to chemicals within the LDW is likely to be less than resident fish species that may spend a substantial portion of their lives in the LDW.

Juvenile chinook are exposed to sediment primarily through their consumption of sediment-associated invertebrates, which are an important prey item in their early estuarine residence (Cordell et al. 1999). Juvenile chinook salmon have been studied in the LDW, and data are available on their exposure within the LDW, as well as potential effects associated with this exposure.

Furthermore, chinook salmon are an icon of the Pacific Northwest. They have been relied on for centuries by indigenous people as a primary food source and are an economic resource of the region as a commercial fishery species. It is likely that some yearling (i.e., fish that have reared for one year in fresh water) juvenile chinook salmon outmigrate through the LDW (Warner and Fritz 1995; Shannon 2001). Yearling chinook tend to occupy deeper water than subyearling chinook and prey mainly on pelagic organisms, including small fish (Healy 1991). Risks to piscivorous yearling juvenile chinook salmon are assumed to be addressed by the bull trout ROC, as discussed below.

A.2.3.2.2 Bull trout

Bull trout, selected as a benthopelagic piscivore, is believed to be exposed to sediment-associated COPCs primarily through its consumption of fish. The Coastal-Puget Sound population of bull trout (*Salvelinus confluentus*) was listed as threatened under ESA on November 1, 1999. Bull trout were selected to represent all piscivorous fish in the LDW for biomagnifying chemicals, such as mercury, DDT and its metabolites (DDTs), and PCBs. This distinction was made because piscivores are likely to have higher body burdens (i.e., higher exposure) of biomagnifying chemicals due to their trophic level, which is higher than fish that consume primarily invertebrates, such as English sole. Other piscivorous (or partially piscivorous) benthic species such as the sand sole or Pacific staghorn sculpin could also have been selected. These species may have greater exposure to sediment-associated COPCs than bull trout because of their close association with sediment and piscivorous diet. However, because no tissue data are currently available for any piscivorous fish, in this Phase 1 risk assessment, bull trout was selected as the ROC to represent piscivorous fish, largely due to its ESA status. The exposure scenario for bull trout was based on conservatively estimated data designed to represent all piscivorous fish. The relative sensitivity of bull trout and other piscivorous fish is unknown, so all tissue-based toxicological data for biomagnifying chemicals will be evaluated (see Sections A.2.4.6 and A.4).

Because bull trout is a threatened species, risks to the piscivorous bull trout from non-biomagnifying compounds, such as arsenic, copper, PAHs, and TBT, are also of concern. Therefore, risks from non-biomagnifying chemicals will be evaluated for bull trout (but not as a representative of other species). Little toxicological research has been conducted on potential effects of these chemicals on bull trout; however, some data for closely related species, such as brook trout and rainbow trout, are available.

Little information is available to characterize use of the LDW by bull trout or other piscivorous fish, such as cutthroat trout, Pacific staghorn sculpin, or sand sole. Bull trout is generally believed to be rare in the LDW. However, by conservatively assuming that bull trout resides in the LDW for its whole life-cycle,[16] bull trout can act as a conservative representative of other piscivorous fish in the LDW for

[16] While bull trout do not reproduce in the LDW, other piscivorous fish do, and therefore, bull trout are assumed to reproduce in the LDW for purposes of evaluating exposures to all piscivorous fish.

biomagnifying chemicals. Because a benthic piscivorous fish such as Pacific staghorn sculpin may have additional sediment exposure, some uncertainty is associated with the assumption that bull trout are the most conservative representative of benthic piscivores. The selection of a piscivorous fish ROC for Phase 2 may differ from bull trout once data gaps are analyzed and the most appropriate fish is identified for field collection. Uncertainties associated with potential exposure to piscivores that are also benthic species will be further discussed in the uncertainty assessment (Section A.7.2.2), and will be addressed through the data gaps process, by collecting and analyzing benthic piscivore tissue.

A.2.3.2.3 English sole

English sole (*Pleuronectes vetulus*) were selected to represent benthic carnivorous fish in the LDW. English sole are a benthic species living in close proximity to sediment, thus giving them a high potential for direct exposure to sediment-associated chemicals. Additionally, English sole feed extensively on sediment-associated invertebrates, and thus are subject to bioaccumulation of sediment-associated chemicals through their diet.

There have probably been more studies conducted on English sole than on any other fish species found in the LDW. A number of studies have examined potential effects of sediment-associated chemicals (e.g., PAHs) on flatfish in the LDW, particularly English sole (e.g., Johnson et al. 1997). Several toxicological studies have used data from English sole collected in the LDW, and tissue concentration data are also available. NMFS data suggest English sole are as sensitive to the effects of PAHs as other flatfish species tested (Myers et al. 1998b). English sole are caught recreationally in the LDW and have some value as a commercial fishery species (though not in the LDW). Except for regionally specific studies conducted with English sole, no preference is given to toxicological data conducted with fish closely related to English sole because the relative sensitivity of other fish represented by this ROC is unknown and may vary widely.

It is assumed that English sole will serve as an appropriate surrogate for other benthic fish species. English sole is one of the most abundant fish in the LDW and is closely related to starry flounder, another of the most abundant fish in the LDW. Exposure and effects studies with English sole should, therefore, be directly relevant to starry flounder.

Exposure of benthic fish such as English sole to sediment-associated chemicals is assumed to be greater than fish with equivalent prey preferences from other habitats. For chemicals that do not biomagnify, exposure of benthic piscivorous fish is assumed to be greater than exposure to pelagic piscivorous fish because of English sole's consumption of prey in direct contact with sediment. In general, sediment-associated organisms preyed on by other fish in the LDW are similar to those preyed on by English sole (see Section A.2.2.3), so their primary exposure route to sediment is similar. Omnivorous fish, such as the shiner surfperch, have additional exposure

routes through consumption of algae and organisms that encrust pilings and other vertical structures. However, because these organisms do not have direct contact with sediment, this exposure route is not likely to result in greater exposure to sediment-associated chemicals than the consumption of benthic invertebrates by English sole (see Tables D-6f, D-6i, and D-6j in Appendix D of the RI). The available information thus indicates assessment of risk for English sole should adequately address risk from all sediment-associated chemicals to fish with omnivorous and carnivorous dietary preferences and fish with benthic, demersal and benthopelagic habitat preferences.

A.2.3.2.4 Summary of fish ROC selection

In summary, three fish species were selected as ROCs to represent risks from sediment-associated chemicals to the fish community of the LDW:

- Juvenile chinook salmon, selected to represent outmigrating juvenile salmonids and juvenile chinook salmon as a threatened species
- Bull trout, selected to represent piscivorous fish for biomagnifying chemicals and bull trout as a threatened species for non-biomagnifying chemicals
- English sole, selected to represent all fish not explicitly represented by the above two ROCs

These fish were selected as ROCs for the Phase 1 ERA because they have the highest potential exposure to sediment-associated chemicals due to their high trophic status (bull trout), and direct contact and benthivorous diet (English sole). Juvenile chinook salmon were selected because they are the most exposed juvenile Pacific salmon and they are a threatened species under the ESA. The piscivorous fish ROC may change in the Phase 2 ERA from bull trout to the species selected for collection and analysis as part of the data gaps process.

A.2.3.3 Wildlife

The potential wildlife receptors of concern were grouped into the following three broad categories based on potential sediment exposure at the site:

- **Piscivorous/carnivorous birds**—including species such as great blue heron, western grebe, cormorant, osprey, and bald eagle
- **Benthivorous birds**—including species such as spotted sandpiper, killdeer, and dabbling ducks
- **Piscivorous mammals**—including species such as river otter and harbor seal

Other broad categories of wildlife receptors, such as herbivorous birds, passerine birds, or omnivorous mammals are assumed to be less exposed to COPCs from the LDW through their diet and sediment ingestion than the three categories listed above. Primarily herbivorous birds, such as geese and some diving ducks, may also feed on benthic invertebrates and may incidentally ingest sediment, but this exposure is assumed to be less than that of benthivorous birds such as shorebirds, which may

ingest significant amounts of sediment while probing intertidal sediment for benthic invertebrates. Ingestion of algae is also a potential exposure pathway. The significance of this pathway depends upon the extent to which sediment-associated chemicals migrate through the water column, are taken up by algae, and then consumed in significant quantities. This pathway is assumed to be insignificant compared to other more direct pathways examined (e.g., incidental ingestion of sediment or contaminated benthic species). Passerine birds are also likely to experience limited exposure to contaminated sediments in the LDW because they primarily use upland habitat. Other mammals, such as raccoons, are expected to have less exposure to sediment-associated chemicals because their food is more terrestrial in origin than the primarily piscivorous river otter and harbor seal.

Using the criteria discussed in Section A.2.3, the following wildlife species were selected as ROCs in the LDW:

- Great blue heron—piscivorous birds
- Bald eagle—piscivorous and carnivorous birds
- Spotted sandpiper—benthivorous birds
- River otter—piscivorous mammals
- Harbor seal—piscivorous mammals

The remainder of this section discusses the rationale for selecting each ROC and how these species will serve as representative species for protection of other similar and important species within the LDW. Species-specific toxicological data were not available for any of the LDW wildlife species to determine which species might be most sensitive to COPCs, although data are available for mink, which are in the same family as river otter.

A.2.3.3.1 Great blue heron

The great blue heron (*Ardea herodias*) was selected to represent the piscivorous bird group because they are year-round residents, known to reproduce and feed in and around the LDW. Additionally, they are susceptible to biomagnification of certain chemicals because of their trophic position and feeding habits. Site-specific data for chemicals in heron food resources are available. It is assumed that great blue heron will serve as a representative species for piscivorous waterfowl and seabirds with similar exposure (i.e., loons, western grebe, mergansers, double-crested cormorant, pigeon guillemot, Caspian tern, common murre).

A.2.3.3.2 Bald eagle

The bald eagle (*Haliaeetus leucocephalus*) was selected to represent piscivorous birds in addition to the great blue heron, as well as carnivorous birds such as peregrine falcon. In addition, the bald eagle was selected because it is listed under ESA as a federally

threatened species, although it is currently under review for delisting. In Washington, it is also listed as a state threatened species (WDFW 2001).

A.2.3.3.3 Spotted sandpiper

The spotted sandpiper (*Actitis macularia*) was chosen to represent the benthivorous bird group because it feeds in the intertidal areas of the LDW from June through September and nests near Kellogg Island and perhaps other areas. Sandpipers feed on invertebrates by probing the sediment, therefore, potentially ingesting significant quantities of sediment in addition to benthic invertebrates. Sandpiper has a higher incidental rate of sediment ingestion (up to 30% of the diet) than other bird species, including ducks and geese (EPA 1993b). It is assumed that because of the high potential exposure through direct ingestion of sediment, the spotted sandpiper will act as a representative species for other benthivorous birds such as scaup and scoters (i.e., diving ducks), as well as geese and dabbling ducks.

A.2.3.3.4 River otter

The river otter (*Lutra canadensis*) was chosen to represent the piscivorous mammal group because otters are suspected to be year-round residents that reproduce and feed in and around the LDW. The river otter is susceptible to biomagnification of chemicals because of its high trophic position and feeding habits. Mustelids are also known to be highly sensitive to certain classes of chemicals, such as PCBs. Site-specific data for chemicals in otter food resources are available, as are relevant toxicological data. Otters also attract a high level of societal interest.

A.2.3.3.5 Harbor seal

The harbor seal (*Phoca vituluna*) was also chosen to represent piscivorous mammals. The harbor seal, like the river otter, is susceptible to biomagnification of chemicals because of its trophic position and feeding habits. Pinnipeds are suspected to be sensitive to certain classes of chemicals, such as PCBs, which may be associated with altered immune function. Site-specific data for chemicals in harbor seal food resources are available. Seals as a group attract a high level of societal interest. It is assumed that the harbor seal will act as a representative species for other marine mammals, such as sea lions or harbor porpoise, that may infrequently use the LDW or have been sighted in Elliott Bay.

A.2.3.4 Plants

Using the criteria discussed above, emergent aquatic plants were selected as an ROC in the LDW. Plants are a potential food source for terrestrial and aquatic animals in the LDW. Plants also provide cover habitat for many fish and invertebrates. Emergent plants are rooted in sediment; thus they are exposed to sediment-associated chemicals directly through root uptake and direct contact, and should have greater sediment exposure than phytoplankton. Some studies on the effects of chemicals on emergent aquatic plants have been published; however, no toxicological data are available

relating sediment chemical concentrations to plant toxicity. The relevance of available soil toxicity data to sediment-exposed vascular plants is somewhat uncertain, but these data are assumed to provide a relevant screen for the purpose of this assessment. Uncertainties regarding use of these toxicity data will be discussed in the uncertainty assessment (Section A.7.4).

A.2.3.5 Summary of ROC Selection

In summary, the following species were selected as ROCs to represent the range of organisms exposed to sediment-associated chemicals in the LDW:

- Benthic invertebrate community
- Crab—higher-trophic-level benthic invertebrate
- Juvenile chinook salmon—anadromous juvenile salmon
- English sole—benthivorous fish
- Bull trout—piscivorous fish
- Great blue heron—piscivorous birds
- Bald eagle—piscivorous/carnivorous birds
- Spotted sandpiper—benthivorous birds
- River otter—piscivorous mammals
- Harbor seal—piscivorous mammals
- Emergent aquatic plants

The selection criteria for each of the above receptors are presented in Table A-2-6 to summarize the rationale for ROC selection.

Table A-2-6. ROCs selected for the LDW and a summary of the rationale for selection

Receptor of Concern	Exposure Route	Ecological Significance	Social Significance	Site Use	Exposure Data Availability	Sensitivity
Benthic invertebrate community	direct contact, diet, sediment ingestion	food source for other invertebrates, fish, and mammals; nutrient cycling	target community for protection in the development of numerical sediment quality criteria	present year-round; multiple life stages	abundant surface sediment data available	due to the diversity of organisms in this ROC group, the range of sensitivities is represented
Crab	direct contact, diet, sediment ingestion	higher trophic level benthic invertebrate	potential human consumption	primarily used by juveniles and adults	site-specific tissue data available	susceptible to bioaccumulation due to trophic position
Bull trout	diet	top of food chain in LDW; preys on other fish	T&E species; previously important sport fish	present at times of high prey abundance (spring/summer)	no tissue data available; prey tissue data available	susceptible to bioaccumulation due to trophic position
English sole	direct contact, diet, sediment ingestion	important prey items for birds and fish; key benthic predator	some recreational and commercial value	juveniles present year round; adults present except when spawning	site-specific fish and prey tissue data available	NMFS data suggest that they are as sensitive as other flatfish (Myers et al. 1998b)
Juvenile chinook salmon	diet	important prey item for birds/fish; seasonally one of the most abundant in the LDW	T&E species; returning adults important to commercial, sport, & tribal fisheries	generally present April-July; most estuary-dependent juvenile salmonid	site-specific fish and prey tissue data available	believed to be sensitive to a wide range of COPCs
Great blue heron	diet, sediment ingestion	high on food chain; preys on fish	charismatic bird	present year-round; reproduce and feed in LDW	site-specific data available for chemicals in some food resources; egg data available	susceptible to bioaccumulation due to trophic position
Bald eagle	diet, sediment ingestion	top of food chain; preys on fish and other small animals	T&E species (under review for delisting)	present year-round; nests in vicinity	site-specific data available for chemicals in some food resources	susceptible to bioaccumulation due to trophic position
Spotted sandpiper	diet, sediment ingestion	preys on invertebrates; important role as an intermediate predator	protected under migratory bird treaty	present June-September; nests along LDW	site-specific data available for chemicals in some food resources	susceptible to bioaccumulation through consumption of invertebrates
River otter	diet, sediment ingestion	top of food chain; preys on fish and crustaceans	charismatic	present year-round	site-specific data available for chemicals in some food resources	susceptible to bioaccumulation due to trophic position; mustelids shown to be highly sensitive to LDW chemicals, e.g., PCBs

Receptor of Concern	Exposure Route	Ecological Significance	Social Significance	Site Use	Exposure Data Availability	Sensitivity
Harbor seal	diet, sediment ingestion	top of food chain; preys on fish	protected under Marine Mammal Act	infrequent	site-specific data available for contaminants in some food resources	pinnipeds suspected to be sensitive to LDW chemicals, e.g., PCBs
Emergent aquatic plants	direct contact	food source for terrestrial and aquatic animals in LDW; provide cover and habitat for a variety of ecological species	important aesthetic concerns; historically important for indigenous cultures' food, basketry, & medicine	present year-round; all life stages present	marsh sediment data available	uncertain; no toxicity data available for estuarine rooted aquatic plants so terrestrial plant toxicity data used

T&E – Species listed as threatened, endangered or sensitive species under the Endangered Species Act.

A.2.4 CHEMICAL OF POTENTIAL CONCERN SELECTION

This section presents the chemical data available for the LDW and provides an evaluation of the relevance of these data to assess exposure of ROCs to sediment-associated chemicals. In addition, through the use of highly conservative, risk-based screens, chemicals are identified as COPCs for each of the ROCs identified in Section A.2.3. If risk is determined to be sufficiently high for a given ROC/COPC pair,[17] additional analysis is conducted and presented in Sections A.3 through A.7. Uncertainties associated with these screens are discussed in Section A.7.

A.2.4.1 Data used in COPC screening

Ecological ROCs are exposed to sediment-associated chemicals found in the LDW primarily either through direct sediment exposure or indirectly through consumption of benthic invertebrates, fish, and shellfish. Accordingly, chemistry data for tissue, sediment, and porewater[18] are relevant. The following sections describe the available data for tissue, sediment, and porewater; the data selection and reduction process; and the data reliability for risk assessment purposes. These analyses are consistent with those conducted for the Phase 1 human health risk assessment for the LDW (Appendix B). A more detailed summary of the data is provided in Section 3.2 of the Phase 1 RI.

Water quality data were not specifically evaluated in this problem formulation because risks attributable to LDW water exposure were evaluated as part of the recent King County Water Quality Assessment (WQA) (King County 1999a,b,c,d). The results of this assessment (discussed in Attachment A.2) indicated that risks to aquatic species in the LDW were low based on a comparison of water quality criteria to measured and modeled[19] chemical concentrations in the water column.

A.2.4.2 Data availability

Environmental investigations conducted within the LDW have included collection and chemical analysis of tissue, sediment, and porewater samples. These data and their respective sources are described briefly below and further in Sections 2.3, 4.1, and 4.2 of the Phase 1 RI.

A.2.4.2.1 Tissue chemistry

Tissue data for the LDW are most abundant for chinook and coho salmon, followed by English sole, mussels, perch, crab, and amphipods (Table A-2-7). Locations of tissue collection are shown in Map A-2-1 (Attachment A.1). PCBs were measured in most

[17] The COPC screen will be verified in the Phase 2 problem formulation using additional data collected through the data gaps process.

[18] Porewater data are not used in this Phase 1 ERA, but may be used in the Phase 2 ERA. They are discussed briefly in this document and also in the Phase 1 RI (Section 4.2.4).

[19] Model was calibrated with field data.

samples. Pesticides and semivolatile organic compounds were also measured frequently. Mercury, methylmercury, arsenic, lead, copper, TBT, and other butyltins were measured in fewer samples. Tissue data in biota collected in the LDW are not available for other chemicals, except for limited PCB congener data. [20] Limitations in available tissue data, including chemicals not previously measured in LDW tissue samples, will be discussed in the uncertainty assessment in Section A.7.

A.2.4.2.2 Sediment chemistry

Approximately 1,200 surface[21] sediment samples have been collected from the LDW within the last 10 years[22] (see Section 2.3.1 in the Phase 1 RI for a complete list of studies). Older data exist, but data quality objectives established in Windward Environmental (2001b) established that data older than 10 years would not be considered representative of current conditions. Intertidal vs. subtidal regions in the LDW are shown in Map A-2-2. For the purposes of this ERA, intertidal locations are assumed to correspond with intertidal habitat identified in USFWS (2000) aerial photo interpretations. The elevation boundary between intertidal and subtidal is approximately -2 ft MLLW. Subtidal stations include all stations that fall within the river boundaries outside of intertidal habitat. Approximately 400 surface sediment samples were collected from intertidal locations;[23] the remainder were collected from subtidal locations.

A.2.4.2.3 Porewater

Limited porewater data are available. In 1997, porewater was collected from 15 stations throughout LDW subtidal areas as part of the EPA Site Inspection of the LDW (Weston 1999). Porewater was analyzed for a total of 28 chemicals.[24] Additional discussion of these porewater data is presented in Sections 2.3.4 and 4.2.4 of the Phase 1 RI.

---

[20] Ylitalo et al. (1999) reported data for 13 PCB congeners in English sole, mussel, and crab collected in Elliott Bay. Crab and English sole collected in Elliott Bay could conceivably receive part of their chemical exposure in the LDW. Also, selected (dioxin-like) PCB congeners were measured in LDW English sole muscle (3 composite samples) and liver (3 composite samples) as part of PSAMP's annual monitoring activities. However, PSAMP has not yet released these data.

[21] For the purposes of this ERA, surface sediment samples are those collected from the top 15 cm of the sediment horizon. Sediment samples that include less than 15 cm of sediment are included; samples that include the top 15 cm, but also include deeper sediment in the same sample are not included here.

[22] Data from the Harbor Island Remedial Investigation were collected more than 10 years ago. For the sake of continuity throughout the project, they are being used in the risk assessment because the data set was identified as a suitable data source at the beginning of the project.

[23] Intertidal locations were sampled during the following events: NOAA SiteChar, Duw/Diag-1, Duw/Diag-2, Norfolk-cleanup1, Norfolk-cleanup2, Boeing SiteChar, and Plant 2 RFI-1

[24] Analytes measured in porewater were arsenic, barium, beryllium, cadmium, calcium, copper, dibutyltin, iron, lead, magnesium, n-butyltin, nickel, potassium, selenium, silver, thallium, tin, tributyltin, vanadium, zinc.

Table A-2-7. Tissue chemistry samples collected from the LDW that were used in Phase 1 risk assessment [a]

Title	Year	Species	N [b]	Number per Composite	Sample Type	Chemicals	Data used in Assessment for:		
							Benthic	Fish	Wildlife
West Waterway Sediment Operable Unit Harbor Island Superfund Site - Assessing human health risks from the consumption of seafood (ESG 1999)	1998	red rock crab	2	5	edible meat		X		
		Dungeness crab	1	1	edible meat		X		
King County Combined Sewer Overflow Water Quality Assessment for the Duwamish River and Elliott Bay (King County 1999a)	1996 - 1997	Dungeness crab	2	3	edible meat	metals, TBT, semivolatiles, PCBs	X	X	X
			1	3	hepatopancreas		X	X	X
		English sole	3	20	skinless fillet			X	
			3	20	whole body			X	X
		amphipods	4	approximately 2,000	whole body			X	X
		shiner surfperch	3	10	whole body			X	X
		mussels	22		whole body				X
Puget Sound Ambient Monitoring Program – annual sampling (West et al. 2001)	1992	English sole	3	5-20	skinless fillet	semivolatiles, pesticides, PCBs, As, Cu, Pb, Hg		X	
	1995	English sole	3	5-20	skinless fillet	pesticides, PCBs, As, Cu, Pb, Hg		X	
	1997	English sole	3	5-20	skinless fillet	Hg, pesticides		X	
Elliott Bay/Duwamish River Fish Tissue Investigation (Battelle Marine Research Laboratory 1996, EVS 1995, Frontier Geosciences 1995)	1995	English sole	3	6	skinless fillet	PCBs, Hg, MeHg, TBT		X	
NMFS Duwamish injury assessment project (NMFS 2002)	2000	chinook salmon (juveniles)	29	1-10	whole body	PCBs, pesticides		X	X
			6	5-10	stomach contents			X	

Title	Year	Species	N[b]	Number per Composite	Sample Type	Chemicals	Data used in Assessment for:		
							Benthic	Fish	Wildlife
Contaminant exposure and associated biochemical effects in outmigrant juvenile chinook salmon from urban and non-urban estuaries of Puget Sound (Varanasi et al. 1993)[c]	1989 - 1990	chinook salmon (juveniles)	14	2-5	whole body	pesticides, PCBs, PAHs		X	X
			6	10	stomach contents			X	

[a] For a complete list of tissue data available for the LDW, see Table 2-5 in the RI.

[b] Most samples were composites of multiple individuals

[c] Six composite samples of juvenile chinook livers were also analyzed, but they were not used in the ERA because toxicity data are not available based on tissue concentrations.

A.2.4.3 Data selection and reduction

This section describes the types of fish tissue or sediment data used in the ERA, because not all data were considered appropriate for use. This section also describes how non-detected, estimated, or duplicate data were treated.

A.2.4.3.1 Tissue chemistry

To screen COPCs for each ROC, it was necessary to compare exposure data to effects data to assess whether a potential for adverse effect exists in the LDW for each ROC/COPC pair. Tissue data were used to estimate exposure in one of two ways: 1) to estimate doses of COPCs to receptors through ingestion of contaminated prey (e.g., for wildlife receptors); and 2) to estimate the integrated exposure of an ROC though the measurement of COPCs in the ROC or surrogate body burdens (e.g., for risks to fish from biomagnifying substances).

For ROC/COPC pairs assessed through dietary exposure, whole-body concentrations were preferred to fillet or organ-specific measurements because ecological receptors generally ingest the majority of the body of their prey (e.g., whole fish, whole invertebrates). Thus, fish data available as cooked skinless fillets, cooked edible meat, or liver were not used in the problem formulation. Uncooked fillet data were used for COPCs without available whole body data where necessary.

For the remaining ROC/COPC pairs, data most closely matching available effects data were preferred. Whole body concentrations are the most common form of available effects data for fish, although some studies report concentrations in other organs (e.g., hepatopancreas) or life stages (e.g., eggs or larvae) correlated with effects (see Section A.4.2). In the following sections, all concentrations in tissue are from whole-body samples unless otherwise stated.

Also, because there is unlikely to be a significant relationship between site-related sediment contamination and tissue concentrations in adult salmon, tissue data for adult salmon were not used in this ERA. Adult salmon are generally believed to feed very little once they re-enter rivers and streams to reach spawning areas. Also, although adult salmon returning to the LDW were exposed to site-related contaminants for a relatively short duration as juveniles during outmigration, the contribution of this short-term exposure to total adult body burdens of COPCs is likely insignificant[25] (O'Neill et al. 1998).

---

[25] For example, a 10-g juvenile chinook salmon with a total PCB concentration of 400 µg/kg (Varanasi et al. 1993) contains 4 µg of PCBs. A 15-kg returning adult chinook salmon captured in the Duwamish River with a total PCB concentration of 50 µg/kg (ESG 1999) contains 750 µg of PCBs, most of which is derived from ingestion of food in Puget Sound and the Pacific Ocean. Thus, even if a lengthy half-life of PCBs is assumed in the fish, less than 1% of the PCB body burden contained in adult salmon could have been obtained from prey items during exposure in the LDW.

Tissue data selected for use in the ERA were utilized in subsequent analyses on an as-reported basis (wet weight [ww]). With the exception of total PCBs and DDTs, a concentration equal to one-half the sample-specific detection limit (as reported by the laboratory) was used for undetected analytes. For total PCBs and DDTs, totals were calculated and stored in the database by summing only detected Aroclors, per SMS rules. In cases where no Aroclor was detected, the detection limit for total PCBs was set equal to the highest detection limit for an individual Aroclor. For exposure calculations, the total PCB concentration based on one or more detected Aroclors was used without modification. For total concentrations based on the highest detection limit for an individual Aroclor, one-half the calculated total PCB concentration was used in exposure calculations. Concentrations generated by the laboratory through duplicate analyses were averaged for use in calculations. All concentrations qualified as estimates (e.g., J) were assumed to be positive identifications and were used as represented in subsequent calculations.

A.2.4.3.2 Sediment chemistry

As described in the sediment data quality objective memorandum (Windward Environmental 2001), some of the surface sediment samples may not reflect current conditions because the sediment previously characterized has been remediated or dredged from the LDW. Section 4.1 in the Phase 1 RI lists the surface sediment samples that were not included in the Phase 1 ERA for this reason. A concentration equal to one-half the sample-specific detection limit was used for undetected analytes in sediment, with the exception of total PCBs and total DDTs, which were calculated as described for tissue in the preceding section. Concentrations generated by the laboratory through duplicate analyses were averaged for use in calculations. All concentrations qualified as estimates (e.g., J) were assumed to be positive identifications and were used as represented in subsequent calculations.

A.2.4.3.3 Porewater

None of the porewater data were specifically excluded.

A.2.4.4 Suitability of data for risk assessment

There are several factors to consider in assessing the suitability and sufficiency of environmental data for risk assessments (EPA 1989, 1990). Of primary importance is the degree to which the data adequately represent site-related contamination, and the expected ecological exposure at the site. Other important considerations are data quality criteria goals, documentation, analytical methods/detection limits, and level of review associated with the data. Because data from many different investigations were available for the LDW, these factors were evaluated for each data set to determine whether it was reasonable to combine these data for use in this ERA.

A.2.4.4.1 Representativeness to site-related contamination and receptor exposure

This section provides an overview of the representativeness of the available tissue, sediment, and porewater data.

Tissue

To be representative, tissue data must provide a reasonable indication of COPC exposure by ROCs at a site. Key considerations in the representativeness of site data are:

- Representativeness of the tissue data with respect to capture location, timing, and home range of the species relative to the site
- Availability of tissue data for COPCs at the site
- Representativeness of tissue data with respect to ROCs at the site and their primary prey items

The home range of fish collected in the LDW may be greater or smaller than the area of the LDW Superfund site. For English sole, for example, considerable uncertainty exists regarding preferred foraging habitat and home range; no site-specific home range estimates have been published for English sole in the LDW. A few home range estimates have been developed using best professional judgment, such as the 9 km^2 home range of English sole, as reported by Puget Sound Dredged Disposal Analysis (PSDDA) (1988). One tagging study (Day 1976) suggests English sole may have some site fidelity, although the "sites" defined in this study are relatively large compared to the LDW. Also, the extent of migration was not established.

When the home range of a particular species does not match the LDW site boundaries, measured body-burdens may over or underestimate contamination associated with the site. It is known that in the winter English sole migrate to Elliott Bay to spawn (Forrester 1969). Also, juvenile chinook salmon pass through the LDW in their migration from either upstream spawning locations or hatcheries. Juvenile chinook salmon released from hatcheries have a small contaminant load before entering the LDW, which is generally attributed to the low levels of some contaminants (e.g., PCBs) found in hatchery feed. Hatchery feed has been found to contain various contaminants (Easton et al. 2002). As such, a portion of their overall contaminant load is not associated with LDW exposure (Meador 2000).

Also, the age of the fish captured can influence the body burden. Older fish tend to have higher concentrations of biomagnifying COPCs in their tissues, and thus their consumption could result in higher exposures to piscivorous receptors. The available English sole and perch data represent adult fish.

Tissue data are not available for all potential receptors and/or associated prey items from the LDW. While it is not necessary to have prey data for all species that inhabit the LDW, site-specific data or the means to estimate COPC concentrations in ROCs or

associated prey are needed for species believed to be most highly exposed to sediment-associated COPCs in the LDW (for dose estimates). Where site-specific data are unavailable, assumptions were required in this ERA to approximate exposure to critical receptors or prey items. These assumptions are discussed in Sections A.4.1 and A.5.1, and also evaluated in the uncertainty assessment (Section A.7). Additional tissue data will be collected and analyzed to fill data gaps identified in the data gaps memorandum to reduce uncertainties in exposure estimates.

Sediment

Many environmental sampling events have included collection of sediment from the LDW (Section 2.3.1 in the Phase 1 RI). The studies have been designed for both reconnaissance (e.g., Boeing SiteChar, EPA SI, and NOAA SiteChar) and focused investigation of suspected areas of contamination (e.g., Boeing RFI, Rhône-Poulenc RFI). The extensive coverage of the reconnaissance surveys, and the focused intensity of facility investigations, indicate available sediment chemistry data are likely representative of the general range of environmental conditions within the LDW. Additional discussion of the distribution of sediment chemistry data and the manner in which they were used in the ERA is provided in the exposure assessment *discussions (Sections A.3.1, A.4.1, A.5.1, and A.6.1).*

Porewater

The porewater data are from 15 locations representing a range of sediment contaminant concentrations (i.e., areas with both high and low contaminant concentrations were sampled), although they may not represent the highest concentrations of certain COPCs.

A.2.4.4.2 QA/QC results

All data sets used in this ERA have been validated by the original study authors or by outside third parties, although the documentation of the data validation or quality review is sometimes minimal. No additional data validation was performed for this ERA. Some results were qualified as unusable[26] by the data validators. Data qualified as unusable were not used in this ERA. Additional data validation may occur during the Phase 2 RI, at which time the suitability of historical data for use in Phase 2 will be determined in consultation with the agencies.

A.2.4.5 Benthic invertebrates

This section presents the COPC screen that was used for benthic invertebrates, including a brief summary[27] of the ecotoxicology of potential COPCs. The results of the COPC screen determine which COPCs were further evaluated for benthic species

[26] Approximately 1,000 results were qualified as unusable out of more than 140,000 analytical results.

[27] The ecotoxicology sections in this problem formulation are not meant to be comprehensive; rather they are intended to provide a brief overview of the types of endpoints generally studied.

in the effects and exposure assessment (Section A.3). A COPC screen for crabs was not conducted in the problem formulation. The COPC screen for crabs is presented in the risk characterization (Section A.7.1) based on the exposure and effects data for crabs presented in Sections A.3.1.2.1 and A.3.2.4.

A.2.4.5.1 Ecotoxicology

Benthic organisms are potentially sensitive to a wide range of chemicals. Most of the toxicity data available are from toxicity tests conducted using field sediment containing multiple contaminants. This section contains a brief discussion of available toxicity data for benthic organisms.

Metals

Toxicity of metals to benthic organisms ranges widely, from a slight reduction in growth rate to mortality. Oligochaetes and mollusks are generally less sensitive to metals (with the exception of TBT) than other aquatic taxa (Leland and Kuwabara 1985). The most sensitive life stages of benthic organisms are the embryonic and larval stages. The speciation and bioavailability of metals determine their relative toxicity. TBT has been observed to cause imposex in snails and suppression of regeneration in echinoderms (Eisler 1989; Gibbs et al. 1990). Mercury adversely affects reproduction, growth, behavior, metabolism, blood chemistry, osmoregulation, and oxygen exchange in benthic organisms (Eisler 1987b).

Pesticides

The mechanisms by which organochlorine pesticides cause toxicity include narcosis (nonspecific toxicity) and more specific mechanisms that result in enhanced toxicity, such as respiratory uncouplers, acetylcholine esterase (AChE) inhibitors, and central nervous system toxicants (Lipnick 1993, McCarty and Mackay 1993).

Relatively little information is available relating sediment-associated pesticides with toxicity to benthic organisms, although some studies with DDT have been conducted (Nebeker et al. 1988). Most sediment guidelines for pesticides have been developed from samples that contain a myriad of other contaminants, any of which may have contributed to the adverse effects observed for those samples.

PAHs

Effects of PAH exposure on benthic invertebrates include inhibited reproduction, delayed emergence, sediment avoidance, and mortality (Eisler 1987a; Landrum et al. 1991). In a study of PAH toxicity to the amphipod *Diporeia*, the mechanism identified as most likely responsible for observed acute toxic responses to PAHs was narcosis (Landrum et al. 1991). Generally, aquatic invertebrates are less able to metabolize PAHs than aquatic vertebrates, although rates of PAH metabolism vary widely within and between phyla (Meador et al. 1995). Thus, invertebrates tend to be more sensitive to PAHs due to acute lethality by narcosis than other organisms that actively metabolize these compounds.

PCBs

Significant interspecies differences in sensitivities to PCBs exist, even among species that are closely related taxonomically (Eisler 1986b). Most studies of the effects of PCBs on benthic invertebrates have evaluated reproductive impairment and effects on survival and growth (Eisler 1986b).

Other Organic Chemicals

Very few data exist on the toxicology of volatile and semi-volatile organic chemicals to benthic organisms. In general, narcosis is the toxic endpoint associated with chemicals such as chlorobenzenes, phthalates, and chlorophenols (EPA 1995; Penttinen and Kukkonen 1998; Fuchsman et al. 1999). Tagatz et al. (1986, as cited in Staples et al. [1997]) provides a study of potential effects of dibutyl phthalate on benthic community structure.

A.2.4.5.2 Screening methods and results

The Statement of Work (SOW) for this RI/FS established the use of numerical chemical standards promulgated under the Washington SMS and relevant benthic tissue effects data to evaluate whether individual chemicals should be retained as COPCs for benthic invertebrates in this ERA. Thus, sediment data described in Sections A.2.4.1 through A.2.4.4 were compared to SQS for all chemicals listed in the SMS.[28] In addition to comparison to SMS, tissue data were evaluated for potential effects of TBT exposure to benthic invertebrates (Section A.3.1.2.2), and were also used to assess risks of COPCs to crabs in Section A.3.1.2.1.

As previously discussed, the SQS were promulgated to address risks to benthic invertebrate communities as a whole, except for higher-trophic-level invertebrates, such as crabs, that may be at greater risk of exposure through bioaccumulation.[29] Application of SQS to predict risks from sediment-associated chemicals to the benthic invertebrate community requires an assessment of both the magnitude and areal coverage of contaminated sediments. SQS values are based on AETs, which are defined as the highest "no effect" chemical concentration above which a significant adverse biological effect always occurred among the several hundred samples used for its derivation. Biological endpoints included in derivation of the SQS chemical standard were field measures of benthic infaunal abundance, and laboratory toxicity tests with marine benthic invertebrate organisms (i.e., amphipods [survival], and oysters [percent abnormal development of oyster larvae]). Representatives of these groups are found throughout the LDW. Under the provisions of the SMS, surface sediments with chemical concentrations equal to or less than all the SQS are

[28] SLs from DMMP were used for chemicals without SQS.

[29] Crab are being evaluated through a tissue approach in Section A.3.1.2, A.3.2.4, and A.7.1.1.2. Note, also, that SMS are not intended to be protective of other receptors (such as fish and wildlife) exposed to sediment-associated COPCs through bioaccumulation. Risks to these receptors are presented in Sections A.7.2 and A.7.3.

designated as having no adverse effects on biological resources (WAC 173-204-310(1)(a)).[30] Note that AETs, which form the basis for the SQS and are discussed further in Section A.3.2.1, were determined using a correlative technique with data from sediment that contains multiple contaminants.

The sediment data described in Section A.2.4.3 were compared to SQS for all chemicals listed in Ecology's SMS. Many SQS values require concentrations normalized to TOC. At very low TOC concentrations, normalization is not appropriate (Michelson and Bragdon-Cook 1993). Concentrations of organic chemicals were not normalized to TOC for samples with TOC concentrations less than or equal to 0.2%. In these cases, dry weight chemical concentrations were compared to the lowest AET, which is functionally equivalent to the SQS. The 0.2% TOC threshold was used similarly by EPA (Weston 1999) in their LDW site inspection. DMMP sediment screening levels (SLs) are available for 16 chemicals for which SMS are not available. A chemical was retained as a COPC if: 1) the maximum detected concentration exceeded the SQS or SL (if an SQS was not available); or 2) the detection limit exceeded SQS or SL (if an SQS was not available), whether or not it was detected elsewhere (Table A-2-8).

No SQS has been developed for TBT. Available screening values include a DMMP porewater (0.15 µg/L) concentration, the acute marine ambient water quality criteria (AWQC) for surface water (0.37 µg/L) and a TBT tissue trigger level (3.0 mg/kg dw) proposed by EPA (1999) for use in evaluating bioaccumulation data from the West Waterway. Using equilibrium partitioning, Weston (1999) calculated sediment concentrations based on the DMMP TBT porewater guideline and AWQC water concentrations. These calculated TBT sediment concentrations for the LDW were compared by Weston (1999) to measured sediment TBT concentrations in the LDW to screen for potential effects to benthic organisms. Based on the screening procedures used by Weston (1999), TBT was retained as a COPC for benthic organisms because measured sediment TBT concentrations in the LDW exceeded sediment concentrations estimated based on the DMMP guidelines (0.15 µg/L in porewater) and AWQC (0.37 µg/L in surface water).

Table A-2-8 lists the chemicals identified as COPCs and retained for further evaluation in the exposure and effects assessment (Section A.3). All chemicals for which SMS are available were retained as COPCs based on the SQS screen. Six of these chemicals were retained because their detection limits were greater than SQS. Based on comparison between maximum detected sediment concentrations in the LDW and the SL, six additional chemicals were identified as COPCs, and are included in Table A-2-8. In addition, eight undetected chemicals were retained as COPCs because

[30] Although designated as such under the provisions of the SMS, due to the SQS derivation process, there is some uncertainty in the prediction of effects based solely on comparison with the SQS.

their detection limits were greater than the SL. A total of 60[31] chemicals were retained as COPCs for benthic invertebrates.

Table A-2-8. Summary of COPCs retained for benthic invertebrates [a,b,c]

COPC	Unit	Max. Conc.	SQS or SL	SQS Ratio [d]
1,2,4-Trichlorobenzene	mg/kg OC	3.6	0.81	4.5
1,2-Dichlorobenzene	mg/kg OC	11	2.3	4.9
1,3-Dichlorobenzene [e]	µg/kg dw	190	170	1.1
1,4-Dichlorobenzene [f]	mg/kg OC	65	3.1	21
2,4-Dimethylphenol	µg/kg dw	290	29	10
2-Methylnaphthalene	mg/kg OC	59	38	1.6
2-Methylphenol [f]	µg/kg dw	2,100	63	33
4-Methylphenol	µg/kg dw	6,250	670	9.3
Acenaphthene	mg/kg OC	170	16	11
Acenaphthylene [e,f]	mg/kg OC	240	66	3.6
Aldrin [e,f]	µg/kg dw	56	10	5.6
Anthracene	mg/kg OC	358	220	1.6
Arsenic	mg/kg dw	150	57	2.6
Benz(a)anthracene	mg/kg OC	808	110	7.3
Benzo(a)pyrene	mg/kg OC	808	99	8.2
Benzo(g,h,i)perylene	mg/kg OC	538	31	17
Benzofluoranthene (total)	mg/kg OC	2,300	230	10
Benzoic acid	µg/kg dw	5,930	650	9.1
Benzyl alcohol	µg/kg dw	1,700	57	30
BEHP	mg/kg OC	520	47	11
Butyl benzyl phthalate	mg/kg OC	540	4.9	110
Cadmium	mg/kg dw	120	5.1	24
Chlordane, alpha [e]	µg/kg dw	26	10	2.6
Chromium	mg/kg dw	1,100	260	4.2
Chrysene	mg/kg OC	808	100	8.1
Copper	mg/kg dw	12,000	390	31
Dibenz(a,h)anthracene	mg/kg OC	277	12	23
Dibenzofuran	mg/kg OC	97	15	6.5
Dieldrin [e]	µg/kg dw	280	10	28
Diethylphthalate [f, g]	µg/kg dw	2,000	48	42
Dimethyl phthalate [f, g]	µg/kg dw	2,000	71	28
Di-n-butyl phthalate [f]	mg/kg OC	310	220	1.4
Di-n-octyl phthalate [f]	mg/kg OC	99	58	4.8
Ethylbenzene [e,f]	µg/kg dw	530	10	53
Fluoranthene	mg/kg OC	2,385	160	15

[31] Sixty chemicals including TBT, which has a porewater-based SL.

COPC	Unit	Max. Conc.	SQS or SL	SQS Ratio [d]
Fluorene	mg/kg OC	169	23	7.4
Gamma-BHC [e,f]	µg/kg dw	56	10	5.6
Heptachlor [e,f]	µg/kg dw	56	10	5.6
Hexachlorobenzene	mg/kg OC	46	0.38	120
Hexachlorobutadiene [f, g]	µg/kg dw	2,000	11	180
Hexachloroethane [e,f]	µg/kg dw	2,100	1,400	1.5
Indeno(1,2,3-cd)pyrene	mg/kg OC	577	34	17
Lead	mg/kg dw	23,000	450	51
Mercury	mg/kg dw	4.6	0.41	11
Naphthalene	mg/kg OC	103	99	1.0
Nickel [d]	mg/kg dw	910	140	6.5
N-Nitrosodiphenylamine [e]	µg/kg dw	2,000	28	71
PCBs (total-calculated)	mg/kg OC	10,600	12	880
Pentachlorophenol	µg/kg dw	540	360	1.5
Phenanthrene	mg/kg OC	1,654	100	17
Phenol	µg/kg dw	3,600	420	8.6
Pyrene	mg/kg OC	1,846	1,000	1.8
Silver	mg/kg dw	270	6.1	44
TBT [b,e]	µg/L	0.080	0.15	0.53
Tetrachloroethene [e,f]	µg/kg dw	536	57	9.4
Trichloroethene [e,f]	µg/kg dw	533	160	3.3
Total DDTs (calculated) [e]	µg/kg dw	2,900	6.9	420
Total HPAH (calculated)	mg/kg OC	9,277	960	9.7
Total LPAH (calculated)	mg/kg OC	2,317	370	6.3
Zinc	mg/kg dw	9,700	410	24

BEHP – bis(2-ethylhexyl)phthalate

na – not available

[a] COPCs retained based on a comparison between maximum sediment concentrations and SMS SQS and DMMP SLs

[b] TBT is also included as a COPC based on equilibrium partitioning analysis conducted according to procedures outlined by Weston (1999), even though the maximum detected concentration in porewater did not exceed the SL

[c] Antimony and xylene were screened out because the maximum detected value or detection limit did not exceed the SL

[d] SQS ratio = maximum measured concentration ÷ SQS; or maximum measured concentration ÷ SL (when SQS is not available). Note that this ratio has no regulatory relevance, and is presented here to indicate the general magnitude of the maximum concentration.

[e] Analyte screened using DMMP SL instead of SQS

[f] Analyte not detected or detected at concentrations less than SQS or SL; detection limit greater than SQS or SL (when SQS is not available)

[g] SQS is in units of mg/kg OC, but the maximum SQS ratio shown is based on a comparison to the lowest AET because TOC normalization was not appropriate for the sample maximum shown. See Section A.2.4.5.2 for additional details.

A.2.4.6 Fish

This section presents the COPC screen for fish ROCs (i.e., juvenile chinook salmon, English sole, and bull trout), including a brief discussion of the ecotoxicology of potential COPCs to fish, covering the range of potential effects reported in the literature.

Risks to juvenile chinook salmon were evaluated in the COPC screen based on potential effects on growth or survival (including reduced survival due to immunosuppression) reported in the literature and other studies. Juvenile chinook salmon were not evaluated for reproductive effects due to their life stage at the time of exposure (i.e., migrating juveniles) and because their exposure to LDW sediments as adults is limited, as previously discussed in Section A.2.4.4. Risks to English sole and bull trout were evaluated based on a comparison of potential exposure in the LDW with toxicity data reported in the literature involving adverse effects to growth, survival, and reproduction.[32]

A.2.4.6.1 Ecotoxicology

Metals

Fish are exposed to metals through their gills and ingestion pathways. Larval stages are generally most sensitive to metal exposure. Certain metals, such as cadmium, lead, copper, and zinc, are more toxic in their free divalent state than in particulate or complexed forms (Wong et al. 1978). Commonly observed effects of metals and metalloids include reduction in growth, survival, and fecundity (Jarvinen and Ankley 1999). Biochemical and histopathological effects have also been reported (e.g., James and Wigham 1986). Mercury can adversely affect fish reproduction, growth, behavior, metabolism, blood chemistry, osmoregulation, and oxygen exchange. Responses to chronic mercury exposure include emaciation, brain lesions, cataracts, diminished responses to change in light intensity, inability to capture food, abnormal motor coordination, erratic behavior, and death (Armstrong 1979; Hawryshyn et al. 1982, both as cited in Eisler 1987b). TBT exposure can result in a variety of adverse effects including inhibition of mitochondrial and oxidative phosphorylation in fish, sluggishness; loss of appetite; altered body pigmentation; air gulping; loss of positive rheotaxis; increased rate of opercular movements; damaged gills, cornea, and epithelial cells of the bile duct; and increases in blood hemoglobin, erythrocyte numbers, and hematocrit (Chliamovitch and Kuhn 1977; Thompson et al. 1985, both as cited in Eisler 1989).

---

[32] In Section A.4.2, only reproductive endpoints relevant to bull trout exposure in the LDW are evaluated for non-biomagnifying chemicals, such as arsenic, copper, TBT, and PAHs. Bull trout do not spawn in the LDW so, for example, egg exposure studies would not be relevant. For biomagnifying chemicals, all reproductive endpoints are evaluated for bull trout, because bull trout serves as an ROC for all piscivorous fish.

Pesticides

Exposure of fish to organochlorine pesticides can result in narcosis (nonspecific toxicity) and more specific toxicity, such as respiratory uncoupling, neuronal acetylcholinesterase inhibition, and central nervous system convulsions (Lipnick 1993; McCarty and Mackay 1993)) . Additionally, the stable DDT metabolites, DDD and DDE, are toxic to a number of fish species. DDT (primarily in the form of DDE) bioaccumulates significantly in fish and other aquatic species. A half-life for DDT elimination from rainbow trout was estimated to be 160 days (EXTOXNET 1996).

Recent research suggests that organophosphate and carbamate insecticides may harm fish by blocking synaptic transmission by inhibiting neuronal acetylcholinesterase (Ferenczy et al. 1997; Sturm et al. 1999), which may result in adverse effects on fish behavior such as predator avoidance and homing behavior. A study by Scholz et al. (2000) reported that the organophosphate pesticide diazinon inhibited olfactory-mediated alarm responses in chinook salmon.

The Washington State Pesticide/ESA Task Force is currently undertaking a systematic evaluation to identify pesticides that may cause harm or are potentially limiting to the recovery of ESA-listed salmonids. This process is evaluating the potential for pesticides to cause direct harm to salmonids, harm through impairment of behavioral patterns, and harm through reduction of prey. Exposure pathways being considered include surface water, diet, sediment, and groundwater intrusion. Results of this evaluation are not complete, and thus were not available for incorporation into this ERA. To date, their research involving pesticides and salmon has focused on water column issues.

PAHs

Both low-molecular-weight polycyclic aromatic hydrocarbons (LPAHs) such as naphthalene, fluorene, phenanthrene, and anthracene and high-molecular-weight polycyclic aromatic hydrocarbons (HPAHs), such as chrysene and benzo(a)pyrene, are acutely toxic to aquatic organisms. Acute lethality increases with increasing alkyl substitution on the lower molecular weight compounds (Van Luik 1984). Many of the HPAHs are also carcinogenic, mutagenic, or teratogenic to a wide variety of organisms including fish (Moore and Ramamoorthy 1984; Eisler 1987a). Exposure to elevated PAH concentrations has been reported to result in reproductive impairment, immune dysfunction, increased incidence of liver lesions, and other histopathological endpoints (Malins et al. 1987; Johnson et al. 1988; Varanasi et al. 1992; Baumann et al. 1996). Fin erosion and liver abnormalities have also been observed in fish exposed to extracts from PAH-contaminated sediments (Fabacher et al. 1991). Other studies report sublethal effects on the cellular immune system (reduced macrophage activities) in fish exposed to PAH-contaminated sediments that could result in increased susceptibility to disease (Weeks and Warinner 1984, 1986; Weeks et al. 1986).

The most common diseases generally affect the liver, although cataracts and disorders of the skin and gills may also occur (O'Connor and Huggett 1988).

In most fish, PAHs are rapidly metabolized and excreted following uptake, as such PAH tissue concentrations are generally low. The major route of elimination is through excretion into bile. Biotransformation and excretion rates can vary widely among fish species (Meador et al. 1995). Fish exposed to PAHs may be induced to produce higher levels of enzymes capable of transforming PAHs to more excretable, but occasionally more toxic, metabolites (O'Connor and Huggett 1988).

PCBs

Effects of PCB exposure on fish include mortality, growth-related impacts, behavioral responses, biochemical alterations, and reproductive impairment. In addition, injection of dioxin-like PCB congeners into fish eggs can cause early-life-stage mortality associated with blue-sac disease, which involves subcutaneous yolk sac edema (Wisk and Cooper 1990; Walker et al. 1991).

Numerous field studies have reported increased mortality, pathologic anomalies, and biochemical changes in feral fish collected from PCB contaminated ecosystems and correlated with tissue concentrations (Niimi 1996). These observations include reduced hatchability and poor survival of larvae taken from feral organisms that were reared in the laboratory (Ankley et al. 1991; Mac and Schwartz 1992). Other effects, such as behavioral responses and biochemical alterations, are more difficult to interpret, although some biochemical alterations may adversely affect reproduction (Sivarajah et al. 1978; Chen et al. 1986; Thomas 1988).

Recent studies have also examined the potential relationship between exposure to contaminants, including PCBs, and increased mortality due to disease by reducing the efficacy of the immune system (Varanasi et al. 1993; Arkoosh et al. 1998b; Powell et al. in press).

Other Organic Compounds

Relatively fewer data exist on the toxicology of other semi-volatile organic compounds to fish. Water toxicity data for volatile organic compounds, such as chlorobenzene, are available, but tissue residue values are not. QSARs (quantitative structure-activity relationships) have been used (Roose and Brinkman 2000) to compare concentrations of volatile organic compounds measured in fish collected from the North Sea to toxicity data for other chemicals. The toxic mechanism of chlorobenzene in fish is narcosis, and the target site is in the cell membrane (Freidig and Hermens 2000).

The mode of toxic action by phenols is thought to be narcosis and/or the uncoupling of oxidative phosphorylation (Penttinen and Kukkonen 1998). Acute toxicity data are available for rainbow trout and fathead minnow (Babich and Stotzky 1985). No chronic toxicity data for phenol were available.

Phthalates have been suggested as a potential endocrine disrupter (Lopes and Furlong 2001). Staples et al. (1997) provides an overview of toxicity studies conducted with fish and water exposures of various phthalates.

A.2.4.6.2 Screening methods and results

This section presents both the methods and results of the COPC screen for fish. As previously indicated, this risk assessment focused primarily on sediment-related pathways. King County (1999c) provided a comprehensive evaluation of risks to fish from surface water in the LDW (risks were found to be generally low; Attachment A.2).

COPCs were screened for fish ROCs using one of two approaches depending on the type of chemical. For most chemicals, a critical tissue residue approach was used as described in detail later in this section. Briefly, using this approach, whole body tissue residues of LDW fish were compared with tissue residue data associated with adverse effects reported in the literature. For most chemicals a critical residue approach approximates the dose at the site of action and eliminates uncertainty associated with uptake and depuration of a chemical. Using the critical tissue residue approach ensures integration of all exposure pathways. For chemicals that are highly metabolized or otherwise regulated by fish such as essential metals and PAHs, a dietary approach was used as described in detail below. For these chemicals the concentration in prey more accurately reflects the toxic dose than do whole-body tissue residues.

COPCs were screened by comparing maximum exposure concentrations in tissue (organism or prey) to no-effect and lowest-effects data. In this document, these data are referred to as no-observed-effects concentrations (NOECs) and lowest-observed-effects concentrations (LOECs) when referring to concentrations in food or whole-body tissues associated with no effects and the lowest effects concentrations, respectively. These data were taken from an EPA toxicity database (AQUIRE), from a recent comprehensive compilation of tissue concentration effects levels (Jarvinin and Ankley 1999), and from the scientific literature as searched through BIOSIS. All original papers were obtained and reviewed for applicability. Studies that satisfy the following data quality criteria were used in these screens:

- The author(s) must report the species and specific chemical information (e.g., "1:1:1:1 mixture of Aroclors 1016, 1221, 1254, and 1260", rather than "mixture of PCBs")
- Only data from single chemical exposures were used, except when the experiment consisted of similar chemicals tested and measured as a mixture (e.g., Aroclor 1254)
- The author(s) must identify the exposure duration associated with the observed effect

- The author(s) must report the chemical concentration or application rate that the fish were exposed to and the associated observed effect
- Biological effects were related to the endpoints of survival, growth, and reproduction
- Concentrations of contaminants in either target organism tissue or its prey were either measured or derived from exposure levels and concentration factors determined in the same study (i.e., no concentrations were estimated using exposure concentration and bioconcentration factors from different studies because of the uncertainty introduced by this approach)

Chemicals were included in the screen if both tissue data and toxicological data for fish were available. Because tissue and toxicological data are limited, a match was not required on a species-specific basis (e.g., trout toxicological data could be compared to perch tissue data from the LDW). Chemicals that were not screened, due to lack of tissue and/or toxicological data, are discussed in the uncertainty assessment (Section A.7.2.2).

Metals

Bull trout, English sole, and juvenile chinook salmon were screened for risk associated with exposure to sediment-associated metals by comparing dietary NOECs and LOECs for growth, mortality, or reproduction to metals tissue residue concentrations in prey. Methods and results are presented below.

NOECs and LOECs were identified for dietary exposure of fish to metals (Table A-2-9). All original papers were obtained and reviewed for applicability. The lowest LOECs and NOECs for each chemical were determined, and these toxicological data were compared with the available LDW tissue data representing prey for the fish ROCs.

In two of the studies reviewed, the chemical was administered via contaminated prey and the measured dry weight concentration in the prey was presented (Hatakeyama and Yasuno 1982; Walsh et al. 1994). In all of the other studies the chemical was added to a commercial fish meal and the nominal or measured fish meal concentration was presented. In general, fish meal is low in moisture (about 10%) so the dietary effects concentrations were assumed to approximate dry weight concentrations. This is a conservative assumption because true dry weight concentrations would likely be somewhat higher than the reported concentrations (see Section A.7.2.2).

Bull trout

The primary route of exposure for bull trout to sediment-associated metals is through food chain transfer from their fish prey.[33] Thus, bull trout were screened by comparing

[33] Bull trout are not believed to ingest a significant quantity of benthic invertebrates in the LDW (see Section A.7.2 for discussion).

the lowest dietary NOECs and LOECs with the highest reported whole-body tissue concentration for fish from the LDW (Table A-2-10). Whole body fish concentrations were converted from wet weight to dry weight assuming 20% solids.

Table A-2-9. Lowest dietary fish LOECs and NOECs for metals

METAL	TEST SPECIES	effect	EFFECTS CONCENTRATION (mg/kg-dw)	REFERENCE
LOEC				
Arsenic	Rainbow trout	growth	30	Oladimeji et al. 1984[a]
Cadmium	Guppy	growth	126	Hatakeyama and Yasuno 1982
Chromium			na	
Copper	Channel catfish	growth	16	Murai et al. 1981
Lead			na	
Silver			na	
Zinc	Rainbow trout	growth	2,000	Takeda and Shimma 1977[b]
NOEC				
Arsenic	Rainbow trout	growth	8	Cockell et al. 1991
Cadmium	Guppy	growth	69.5	Hatakeyama and Yasuno 1982
Chromium	Thick-lipped gray mullet	growth	9.42	Walsh et al. 1994[c]
Copper	Channel catfish	growth	8	Murai et al. 1981
Lead	Rainbow trout	growth	7,040	Goettle et al. 1976
Silver	Rainbow trout	growth	3,000	Galvez and Wood 1999
Zinc	Rainbow trout	growth	1,000	Takeda and Shimma 1977

na – not available

[a] Concentrations in a figure and in the text of this reference do not agree (20 mg/kg is mentioned both as an effect level and a NOEC in the text). However, it is shown in the figure to be non-significant (i.e., a NOEC). In this ERA, the 20 mg/kg-diet exposure is thus assumed to be a NOEC. Concentrations are reported as dry weight

[b] Fish fed 0.01% Ca in diet. Fish fed at same dose Zn with 0.5% Ca experienced no adverse effects.

[c] There was actually a significant increase in growth of the Cr-exposed fish (not an adverse effect). Fish were exposed to Cr in both diet and sediments simultaneously.

Table A-2-10. Bull trout metals screen

Metal	Maximum Fish Tissue Concentration (mg/kg dw)	Species Measured[a]	LOEC[b] (mg/kg dw)	NOEC[b] (mg/kg dw)
Arsenic	26	English sole	30	8
Cadmium	0.10[c]	shiner surfperch	126	69.5
Chromium	1.5	English sole	na	9.42
Copper	11[c]	shiner surfperch	16	8
Lead	0.84	English sole	na	7,040
Silver	<0.054	English sole	na	3,000
Zinc	95[c]	shiner surfperch	2,000	1,000[d]

na – Not available

[a] Species in which maximum tissue concentration from LDW was measured

[b] TRVs are described further in Table A-2-9

[c] Concentration converted from wet weight assuming 20% solids

Fish tissue concentrations of arsenic and copper were greater than their respective NOECs. Therefore, arsenic and copper were retained as COPCs for bull trout in the fish effects and exposure assessment (Section A.4) and risk characterization (Section A.7.2), and cadmium, chromium, lead, silver, and zinc were assumed to pose negligible risk.

Juvenile chinook salmon and English sole

Juvenile chinook salmon and English sole were screened by comparing maximum modeled amphipod tissue concentrations (Table A-2-11) to the lowest dietary NOECs and LOECs. Epibenthic invertebrates, such as amphipods, make up a large portion of the diet for both juvenile chinook salmon and English sole (Section A.2.2.3). Amphipods live in and on sediment and are detritus feeders or scavengers that feed by filtering water or sediment through their appendages. These life history characteristics result in assimilation of sediment-associated chemicals that is likely similar to other epibenthic prey species in the LDW (Section A.7.2.2).

Amphipod tissue data[34] were available from two sites in the vicinity of Kellogg Island. To estimate amphipod tissue metals concentrations at locations with higher sediment metals concentrations within the LDW, a site-specific biota accumulation factor ($BAF = C_{tissue} \div C_{sed}$)[35] was calculated. Non-lipid- or TOC-normalized BAFs are commonly used to relate metal concentrations in benthic invertebrate tissues to concentrations in sediment in a site-specific manner (e.g., Thomann et al. 1995; Bechtel Jacobs 1998).

[34] Four composite samples consisting of approximately 87% *Eogammarus* (an epibenthic amphipod) and 13% *Corophium* (an infaunal amphipod) (King Co 1999).

[35] Note that the BAF for metals is not organic carbon- or lipid-normalized.

BAFs were calculated using synoptic sediment data collected from the vicinity where the amphipods were collected. Sediments were collected from three stations near Kellogg Island and corresponded to two amphipod composite samples. In addition, sediments were collected from one station on the West Marginal Way side of the channel adjacent to Kellogg Island, corresponding to two additional amphipod composite samples, for a total of four composite samples. Amphipod dry weight tissue concentrations were based on a measured 17.9% solids content of the Kellogg Island samples. A BAF was calculated for each of the two collection sites based on the average amphipod tissue concentration and the average sediment concentration from each site.

The highest BAF was then applied to the 95th percentile surface sediment concentration for each metal from all samples collected in the LDW (Table A-2-11). This approach likely provides a conservative estimate of the metal concentrations in amphipods, because uptake is probably not a linear function of sediment metals

Table A-2-11. Comparison of estimated amphipod tissue concentrations in LDW to toxicity data

Analyte	Avg KI Amphipod (mg/kg dw)	Avg KI Sediment (mg/kg dw)	BAF KI	Avg WMW Amphipod (mg/kg dw)	Avg WMW Sediment (mg/kg dw)	BAF WMW	95th Percentile of LDW Sediments (mg/kg dw)	Estimated Amphipod Tissue (mg/kg dw)	Lowest LOEC (mg/kg dw)	Lowest NOEC (mg/kg dw)
As	5.5	5.7	0.96	7.7	12	0.64	28	27	30	12
Cd	0.11	0.28	0.39	0.52	0.16	3.3	2.0	6.6	126	69.5
Cr	3.0	16	0.19	2.7	19	0.14	64	12	na	9.4
Cu	61	20	3.0	150	65	2.3	130	390	16	5.3
Pb	6.3	17	0.37	35	110	0.32	200	74	na	7,040
Ag	0.39	0.14	2.8	0.50	0.22	2.3	1.8	5.0	na	3,000
Zn	48	45	1.1	10	120	0.83	370	410	2,000	1,000

KI – Kellogg Island

WMW – West Marginal Way

BAF – bioaccumulation factor

concentration over the concentration range of metals in the LDW (i.e., BAFs tend to decrease with increasing metal concentrations in sediment).

The estimated amphipod tissue concentrations calculated using the highest BAF (Table A-2-11) were compared with dietary NOECs and LOECs for each metal for which both amphipod data and toxicological data were available.[36] Estimated

[36] Note that concentrations of these chemicals in soft tissues of mussels and crabs (edible meat, as well as predicted whole body including hepatopancreas data) collected from the LDW were all lower than

amphipod tissue concentrations of arsenic and copper exceeded the respective NOECs, and thus these metals were retained as COPCs in the effects and exposure assessment (Section A.4) and risk characterization (Section A.7.2) for juvenile salmonids and English sole. Cadmium, chromium, lead, silver, and zinc were assumed to pose negligible risk.

Although the estimated amphipod concentration for chromium (12 mg/kg) exceeded the single NOEC available for chromium, the use of this NOEC is highly conservative. Chromium is an essential element, and experimental fish exposed at 9.4 mg Cr/kg diet (dw) actually grew significantly more than control fish ($p<0.01$) (Walsh et al. 1994). Based on this study, it is unlikely that chromium exposure in the LDW is at concentrations likely to cause adverse impacts to fish. Thus, chromium was not retained as a COPC for juvenile chinook salmon or English sole.

Mercury

Fish were screened for adverse effects of mercury by comparing the highest whole-body tissue concentrations reported for fish collected from the LDW to the lowest whole-body (including egg and embryo) tissue NOECs and LOECs associated with adverse effects to growth, mortality or reproduction. LDW tissue concentration data were available for English sole and shiner surfperch.[37] The highest whole-body mercury concentration from either fish was selected for comparison (i.e., 0.088 mg/kg ww in shiner surfperch).[38] Because this concentration was higher than the lowest LOEC reported in the literature (0.036 mg/kg ww in embryos associated with reduced larval rainbow trout growth [Birge et al. 1977]), mercury was retained as a COPC for English sole, bull trout, and juvenile chinook salmon.

TBT

Fish were screened for adverse effects of exposure to TBT by comparing maximum tissue concentrations reported for fish collected from the LDW to the lowest whole-body tissue NOEC and LOEC associated with adverse effects to growth, mortality or

---

predicted amphipod concentrations except for arsenic in crab (average 47 to 49 mg/kg dw and max 60 to 61 mg/kg dw for edible meat and predicted whole body, respectively) (see Tables D-6f, D-6i, and D-6j in Appendix D of the RI for summary statistics of crab, amphipod, and mussel tissue data, respectively). Thus, use of these mussel or crab data, except in the case of arsenic, which is retained as a COPC based on modeled amphipod data, would have provided a less conservative screen than that presented. Additionally, note that for all chemicals screened out, except chromium, the 95th percentile sediment concentration is lower than the lowest NOEC; thus, inclusion of sediment ingestion would not have changed the screen. See text regarding chromium NOEC. Sediment ingestion is discussed further in the uncertainty assessment (Section A.7.2.2).

[37] Hatchery and wild juvenile chinook salmon collected near Kellogg Island in June 2002 were analyzed for mercury, PCBs, lipids, and moisture. These data will be available for use in the data gaps process and Phase 2.

[38] Because of their shorter residence time in the LDW, concentrations in juvenile chinook salmon are assumed to be less than those measured in shiner surfperch or English sole, and thus the lack of data for juvenile chinook should not impact the conservative nature of this screen.

reproduction. Tissue concentration data were available for English sole and shiner surfperch. The highest whole-body TBT concentration from either fish was selected for comparison (i.e., 0.18 mg/kg ww in shiner surfperch). Because TBT does not biomagnify, concentrations in shiner surfperch or English sole were assumed to be representative of those in bull trout and no biomagnification factor was applied.

The lowest LOEC identified in the literature was 1.79 mg TBT ion/kg ww in a male saltwater goby (Shimizu and Kimura 1987), in which the goby was exposed to tributyltin oxide (TBTO) and observed effects on gonadal development were monitored. TBT ion concentration was converted from TBTO using a conversion factor of 0.97 based on the molecular weight of bis(tributyltin)oxide relative to the molecular weight of the TBT ion.

Because no NOEC was available, the NOEC was estimated by dividing the LOEC by a factor of 10 (EPA 1994). Because the maximum TBT tissue concentration in fish collected in the LDW (0.18 mg/kg ww) was equal to the LOEC/10 (0.18 mg/kg ww), TBT was retained as a COPC for all three fish ROCs in the effects and exposure assessment (Section A.4) and risk characterization (Section A.7.2).

Pesticides

Fish were screened for pesticide COPCs by comparing the measured or estimated maximum whole body tissue concentration reported for fish collected from the LDW to the lowest whole body (including egg and embryo) tissue NOEC and LOEC reported in the literature to be associated with adverse effects to growth, mortality, or reproduction (Table A-2-12).

Pesticides have been measured in whole body samples of juvenile chinook salmon and fillet samples[39] of English sole. Concentrations in whole body bull trout were estimated using a predator-prey factor (PPF) of 3.5 based on a PCB study in the Great Lakes (Metcalfe and Metcalfe 1997) because no PPF for DDT was found. A PPF was used because no piscivorous fish tissue data were available. PPFs account for biomagnification through the food chain. Uncertainties due to use of a PPF derived for PCBs to estimate DDT tissue concentrations and regarding use of fillet data are discussed in the uncertainty assessment (Section A.7.2.2).

Because the maximum DDT concentration measured in juvenile chinook salmon was greater than the lowest NOEC (Table A-2-12), DDT was selected as a COPC for

[39] Fillet samples were used for DDT because no whole-body tissue data were available and stakeholders requested assessment of concentrations in fillet samples for DDT.

Table A-2-12. Comparison of LDW chinook and English sole whole body tissue concentrations of organochlorine pesticides with lowest reported NOECs and LOECs

Chemical	Lowest LOEC (mg/kg ww)[a]	Endpoint	Test Species	Reference	Lowest NOEC (mg/kg ww)[a]	Endpoint	Test Species	Reference	Max. Chinook Tissue Conc. (mg/kg ww)	Max English Sole Fillet Tissue Conc. (mg/kg ww)	Max Estimated Bull Trout Tissue Conc. (mg/kg ww)[c]
DDT[d]	1.27	reproduction	rainbow trout	Hopkins et al. 1969	0.025	survival	Golden shiner	Courtney and Reed 1971	0.049	0.011	0.17
Dieldrin	1.21	LR98	winter flounder (embryo)	Smith and Cole 1973	0.36	growth	rainbow trout (juvenile)	Shubat and Curtis 1986	0.002	<0.001[b]	0.007
Heptachlor	11.5	Survival reduced	spot (saltwater juvenile)	Schimmel et al. 1976	2.9	survival	spot (saltwater juvenile)	Schimmel et al. 1976	<0.0003[b]	<0.0005[b]	nd
Lindane	79	LR50	sheepshead minnow (17-21 mm)	Schimmel et al. 1977	na	na	na	na	<0.0004[b]	na	nd

[a] NOECs and LOECs were reported on a whole body basis.

[b] Non-detect. Detection limits for the original samples are presented.

[c] Based on a PPF of 3.5 applied to juvenile chinook whole body tissue concentrations.

[d] Residues are sum of detected DDT and its metabolites (DDD and DDE) measured in fish tissue.

LR98 – statistically determined tissue residue at which 98% mortality occurs

LR50 - statistically determined tissue residue at which 50% mortality occurs

na - not available or applicable

nd - not determined because it was not detected in juvenile chinook salmon whole body tissue residues

juvenile chinook salmon. Based on the chinook data multiplied by a PPF of 3.5, DDT was also selected as a COPC for bull trout. None of the concentrations of pesticides in English sole fillet samples were greater than their respective NOECs. However, because whole-body concentrations are generally higher than fillet tissue concentrations and the fillet concentration is close to the NOEC (within a factor of 3), DDT was included as a COPC for English sole based on uncertainty in the available tissue residue data. Pesticides with very little (e.g., lindane) or no (e.g., aldrin) toxicity data are discussed in the uncertainty section (Section A.7.2.2).PAHs and PCBs

Juvenile chinook salmon

Elevated PCB concentrations in juvenile chinook salmon livers and stomach contents, elevated PAH concentrations in stomach contents, and elevated concentrations of fluorescent aromatic compounds (FACs) in bile were measured in juvenile chinook collected in the LDW (McCain et al. 1990; Varanasi et al. 1993; Stein et al. 1995; Collier et al. 1998). A series of studies have been conducted to assess the potential effects of PCB and PAH exposure on juvenile chinook (Arkoosh et al. 1991; Varanasi et al. 1993; Arkoosh et al. 1998a). These studies examined growth, immunocompetence, and biochemical alterations in field-collected fish. In addition, several studies examined effects in fish following injection with PAHs, PCBs, or sediment extracts containing these and other chemicals from the Hylebos Waterway in Tacoma, Washington (Arkoosh et al. 1994; Arkoosh et al. 1998c; Casillas et al. 1998a,b). In addition, two recent studies have examined the potential of PCB- and PAH-spiked food to impact the survival, growth, and immunocompetence of juvenile chinook salmon (Powell et al. in press; Palm et al. in prep). Due to the abundance of data available regarding survival and growth endpoints, and the need for careful evaluation of all results, PCBs and PAHs were screened in for further evaluation in the effects and exposure assessments (Section A.4) and risk characterization (Section A.7.2) for juvenile chinook salmon.

English sole

Exposure of flatfish to PAH-contaminated sediment has been linked to some adverse effects, including reproductive impairment, DNA damage, and impaired growth (Johnson et al. 2002). PAH exposure is associated with liver lesions and other histopathological changes (Malins et al. 1987; Johnson et al. 1988; Varanasi et al. 1992; Baumann et al. 1996). English sole collected in the LDW have been reported to exhibit reproductive dysfunction relative to reference sites (Johnson et al. 1997) and to have a higher prevalence of liver lesions than reference populations (O'Neill et al. 1998). Due to the abundance of site-specific data regarding survival, growth, and reproductive endpoints, PAHs were retained as a COPC for further analysis for English sole in the effects and exposure assessment and risk characterization (Sections A.4 and A.7.2).

PCBs were assessed for English sole by comparing the highest tissue concentration in English sole to the lowest whole body NOECs and LOECs reported for adverse effects

to growth, mortality, and reproduction in fish. English sole tissue concentrations were higher than the NOEC for Aroclor 1254 (Hansen et al. 1973) and the LOEC for Aroclor 1260 based on a study with fathead minnow (van Wezel et al. 1995) (Table A-2-13). Based on this assessment, PCBs were retained as a COPC for English sole in Sections A.4 and A.7.2.

Table A-2-13. Comparison of LDW shiner surfperch and English sole whole-body tissue concentrations of PCBs (mg/kg ww) with lowest reported NOECs and LOECs

	Effects Conc.	Species	Endpoint	Reference	Maximum Shiner Surfperch PCB Tissue Concentration	Maximum English Sole PCB Tissue Concentration	Estimated Bull Trout Tissue Conc.[a]
LOEC							
Aroclor 1260	0.36	Fathead minnow, 6 months	Reduced survival, death	van Wezel et al. 1995	0.25	0.61	0.88
NOEC							
Aroclor 1254	0.26	Sheepshead minnow larvae	Survival	Hansen et al. 1973	0.37	0.75	1.3

[a] Calculated using a PPF of 3.5 applied to maximum whole body shiner surfperch concentration

Bull trout

Risks from PCBs to bull trout were assessed by comparing the highest estimated tissue concentration of a given Aroclor in LDW bull trout[40] to the lowest NOECs and LOECs for growth, mortality, and reproduction in fish. The estimated bull trout tissue concentrations were higher than the LOEC for Aroclor 1260 (Table A-2-13), and thus PCBs were retained as a COPC for bull trout.

With respect to risks from PAHs to bull trout, the most significant potential exposure route for hydrophobic organic compounds to bull trout is through their diet. Because fish, the primary component of the bull trout diet, rapidly metabolize PAHs (Varanasi et al. 1989), the bull trout diet is expected to contain very low concentrations of PAHs or their metabolites, and thus the exposure of bull trout to PAHs is limited[41]. Based on the likely lack of a complete exposure pathway, as well as the low probability for risk

[40] Based on tissue concentrations of a given PCB Aroclor in shiner surfperch and a PPF of 3.5 (based on Metcalfe and Metcalfe [1997])

[41] A worst-case scenario estimate was calculated to evaluate potential PAH exposure to bull trout if benthic invertebrates were consumed. Potential bull trout PAH exposure was calculated assuming 100% consumption of estimated PAH concentrations in amphipods from the most contaminated site in the LDW. The resulting estimated maximum dietary amphipod concentration was 61 mg/kg (dw). This value was lower than the NOEC of 100 mg/kg-diet (dw) for growth of rainbow trout (Hart and Heddle 1991), indicating that risk to bull trout from ingestion of amphipods in the LDW is low. For details of this analysis see Section A.7.2.2.1.

even if the pathway were present (see Section A.7.2.2.1), PAHs were not retained as a COPC for bull trout, and this pair is not evaluated further in Sections A.4 and A.7.2.

Other organic compounds

A search was conducted for toxicological data for organic compounds other than pesticides, PAHs, and PCBs, to compare to whole body tissue concentrations measured in LDW fish. Data for 13 additional chemicals were located, and their corresponding lowest NOECs and LOECs were compared to perch data, the only fish in which these chemicals were measured in whole body tissues (Table A-2-14).[42] None of these chemicals were detected in perch tissue, and except for the chemicals discussed below, all of the detection limits were lower than the NOECs for these chemicals, so they were not retained as COPCs.

Pentachlorophenol, hexachlorocyclopentadiene, and hexachloroethane were not retained as COPCs because they were undetected at a detection limit greater than or equal to the NOEC, and thus they are unlikely to be present at a concentration greater than the NOEC. Additionally, the only NOEC available to screen hexachloroethane was from a bioconcentration study that was not explicitly designed to assess toxicity (Oliver and Niimi 1983). Compounds that could not be adequately screened due to a lack of toxicological or tissue data are discussed further in the uncertainty assessment (Section A.7.2.2).

In summary, COPCs retained for fish ROCs include arsenic, copper, TBT, mercury, PCBs, and DDT for all three fish ROCs. PAHs were also selected as a COPC for juvenile chinook salmon and English sole.

[42] Fish tissue data are further described in Section A.2.4.2.1

Table A-2-14. Comparison of LDW perch whole-body tissue concentrations of other organic chemicals with relevant effects concentrations

Chemical	LOEC (mg/kg ww)	Endpoint	Test Species	Reference	NOEC (mg/kg ww)	Endpoint	Test Species	Reference	Max. Tissue Conc. in LDW Perch (mg/kg ww)
1,2,4-trichlorobenzene	182	survival	fathead minnow (6 mo)	van Wezel et al. 1995	0.18	survival	RBT (subadult 250 g)	Oliver and Niimi 1983	<0.024
1,2- dichlorobenzene	138.2	survival	RBT (6 mo)	van Wezel et al. 1995	0.67	survival	RBT (subadult 250 g)	Oliver and Niimi 1983	<0.024
1,3- dichlorobenzene	170	growth	fathead minnow (embryo-juvenile)	Carson and Kosian 1987	0.64	survival	fathead minnow (subadult 250 g)	Oliver and Niimi 1983	<0.024
1,4- dichlorobenzene	103	growth	fathead minnow (embryo-juvenile)	Carson and Kosian 1987	0.54	survival	RBT (subadult 250 g)	Oliver and Niimi 1983	<0.024
bis(2-ethylhexyl)phthalate	1.5	survival	(egg – juvenile)	Mehrle and Mayer 1976 [a]	0.39	survival	RBT (eggs – juvenile)	Mehrle and Mayer 1976 [a]	<0.24
Hexachlorobenzene	na				0.16	survival	RBT (subadult 250 g)	Oliver and Niimi 1983	<0.04
Hexachlorobutadiene	na				0.06	survival	RBT (subadult 250 g)	Oliver and Niimi 1983	<0.04
Hexachlorocyclopentadiene	0.08	survival	fathead minnow	Spehar et al. 1977	0.04	survival	RBT (subadult 250 g)	Spehar et al. 1977	<0.04
Hexachloroethane	na				0.0071	survival	RBT (subadult 250 g)	Oliver and Niimi 1983	<0.04
Phenol	79	survival	goldfish (2.2 g)	Kishino and Kobayashi 1995	25	survival	goldfish (2.2 g)	Kishino and Kobayashi 1995	<0.16
Pentachlorophenol	22.1	growth	fathead minnow (larvae-juvenile)	Spehar et al. 1985	0.04	survival	RBT (40 g)	Niimi and Cho 1983	<0.04
2-chlorophenol	128	survival	goldfish (2 g)	Kobayashi et al. 1979	50	survival	goldfish (2.2 g)	Kishino and Kobayashi 1995	<0.08
4-nitrophenol	95	LR50	sheepshead minnow (juvenile)	Brecken-Folse et al. 1994	25.1	survival/ growth	fathead minnow (juvenile)	Call et al. 1980	<0.08

[a] Tissue residue calculated from BAF presented in this study.

na – not available

LR50 – statistically determined tissue residue at which 50% mortality occurs

RBT – rainbow trout

A.2.4.7 Wildlife

This section presents the COPC screen for avian and mammalian wildlife, including a brief discussion of COPC-specific ecotoxicology. The screen is largely based on the results from King County's detailed wildlife assessment (King County 1999), which is discussed in this section. In addition to the King County wildlife results, further screening was conducted for PCBs, DDT, and mercury, and results are presented in Section A.2.4.7.2. The results of these screens determine the ROC/COPC pairs to be further evaluated in the wildlife exposure and effects assessment and risk characterization (Sections A.5 and A.7.3).

A.2.4.7.1 Ecotoxicology

This section provides a brief overview of potential adverse ecological effects to birds and mammals associated with exposure to the various groups of chemicals present in the LDW.

Metals

Birds

Avian dietary toxicity studies have been conducted with a wide range of metals. Sublethal effects can include reproductive and behavioral modifications. Teratogenic effects have been documented in chicken embryos after eggs were injected with chromium (Ridgeway and Karnofsky 1952; Gilani and Marano 1979, as cited in Eisler 1986). Methylmercury tends to be more toxic to birds than inorganic mercury, and young birds are more sensitive than older birds (Eisler 1987b). Sublethal mercury poisoning can cause adverse effects on growth, development and reproduction, blood and tissue chemistry, metabolism, and behavior. Muscular incoordination, slowness, withdrawal, and hypoactivity have been observed in birds exposed to mercury (Eisler 1987b). Triorganotins are considered to be the most toxic of the organic-tin compounds. Possible effects of triorganotin poisoning include tremors, ataxia, lethargy, and degeneration and necrosis of the large neurons of the pons, medulla oblongata, gray matter of the spinal cord, and cells of the cerebral cortex (Eisler 1989). Embryotoxic effects have been observed from subchronic dietary exposure of quail to TBT in laboratory studies (Schlatterer et al. 1993; Coenen et al. 1992).

Mammals

Methylmercury can biomagnify within food chains and result in greater exposure of higher trophic level wildlife in aquatic systems. Organomercury compounds, especially methylmercury, are the most toxic form of mercury to mammals. Mercury causes teratogenic, mutagenic, and carcinogenic effects in mammals. The kidney is the primary organ affected by mercury poisoning in adult mammals, and the brain is the primary target organ in fetuses (Suzuki 1979; Khera 1979, both as cited in Eisler 1987b). At low concentrations, mercury can affect reproduction, growth and development, behavior, blood and serum chemistry, motor coordination, vision, hearing, histology,

and metabolism (Eisler 1987b). Larger mammals such as seals appear to be more resistant to mercury than smaller mammals such as mink and river otters (Eisler 1987b). The reasons for these differences in sensitivity are unknown, but may be related to differences in metabolism and detoxification. Lead modifies the function of and structure of kidney, bone, the central nervous system, and the hematopoietic system, and produces adverse biochemical, histopathological, neuropsychological, fetotoxic, teratogenic, and reproductive effects (Eisler 1988a). TBT is highly toxic to mammals. It causes chromosomal aberrations and reduction in thymus weight (Snoeij et al. 1985; Dixon and Prosser 1986, both as cited in Eisler 1989).

Pesticides

Birds

Birds are generally less sensitive to dieldrin than aquatic organisms, although exposure may be greater because they are higher in the food chain and dieldrin can biomagnify. Gamma-BHC is slightly to moderately toxic to birds; eggshell thinning and reduced egg production have occurred in birds exposed to gamma-BHC (EXTOXNET 1996).

There has been much concern over chronic exposure of bird species to DDT and its metabolites (DDD and DDE) and associated effects on reproduction, especially eggshell thinning and embryo mortality. The mechanism associated with eggshell thinning is not fully understood, although it is believed that predatory birds may be more sensitive to these effects. Laboratory studies on avian reproduction have demonstrated the potential for DDT and DDE to cause subtle changes in courtship behavior, delays in pairing and egg laying, and decreases in egg weight in ring doves and Bengalese finches (EXTOXNET 1996).

Mammals

Some organochlorine pesticides such as o,p'-DDT, kepone, and methoxychlor have estrogenic activity in wildlife. Many of these compounds, including o,p'-DDT and kepone, have been shown to act by binding to the estrogen receptor. However, other organochlorine compounds can exert estrogenic or anti-estrogenic effects by other mechanisms (Carey et al. 1998). The overall impact of such estrogenic activity is typically disruption of normal reproductive functioning.

In addition, several chlorinated pesticides are known to affect mammalian immune system function. These pesticides include hexachlorobenzene, mirex, lindane, chlordane, dieldrin, and DDT and its metabolites (Carey 1994). The immunotoxic effects of these compounds have been demonstrated in several species and include loss of resistance to infections. In most cases, the mechanism of action for these compounds is not well known.

PAHs

Birds

Very few data are available on the toxicity of PAHs in birds. In one study, Patton and Dieter (1980) fed mallards a diet spiked with PAHs for a period of 7 months. No mortality or visible signs of toxicity were evident during the exposure; however, liver weight increased 25%, and blood flow to the liver increased 30% when compared to controls (Eisler 1985). In addition, PAH mixtures applied to the surface of mallard eggs have been shown to result in increased embryo mortality and increased embryo deformation (Hoffman and Gay 1981).

Mammals

In mammals, several PAHs have been shown to be potent carcinogens. In general, carcinogenic PAHs transform cells through genetic injury involving metabolism of the parent compound to a reactive diol epoxide (Eisler 1985). In the case of benzo(a)pyrene, one isomer of the 7,8-diol, 9,10-epoxide is an exceptionally potent carcinogen to newborn mice and is believed to be the ultimate carcinogenic metabolite of this PAH (Slaga et al. 1978). One of the most toxicologically significant processes involved in response to PAH exposure is the interaction with drug-metabolizing enzyme systems. Increased production of mixed-function oxidase enzymes in various small mammals has been induced by numerous PAH compounds (EPA 1980). Interspecies differences in sensitivity to PAH-induced carcinogenesis are due largely to differences in levels of mixed-function oxidase activities that affect rates at which active metabolites are converted to less active products (Neff 1979).

PCBs

Birds

Chronic dietary exposure of various bird species to PCBs has been reported to result in various reproductive effects, including reduced hatching success, fledging rate, and egg production; embryo mortality; developmental deformities; and altered parenting behavior (Peakall 1986; Giesy et al. 1994a; Barron et al. 1995; Hoffman et al. 1996). In addition, there appears to be significant inter-species variability in avian sensitivity to PCBs.

The most sensitive avian species tested in the laboratory appears to be domestic chickens, based on work done by Scott et al. (1971), Britton and Huston (1972, 1973), Lillie et al. (1974, 1975), and Ax and Hansen (1975). The other avian species for which extensive laboratory testing has been conducted is the mallard duck (Heath et al. 1972; Custer and Heinz 1980), which appears to be less sensitive than the domestic chicken. Controlled dietary exposures to PCBs have been conducted for a few other bird species (e.g., bobwhite quail, screech owls, pheasants), though few studies have described complete exposure-response relationships, most consisting of a single dietary dose.

Potential adverse reproductive and developmental effects in wild, piscivorous bird populations exposed to PCBs has been the subject of numerous studies (Tillitt et al. 1992; Jones et al. 1993, 1994; Giesy et al. 1994a,b). Much of this research has focused on Great Lakes populations of double-crested cormorants, because reduced reproductive success and deformities in this species were found to coincide with high exposure to organic pollutants, including PCBs. In addition to embryo mortality, PCBs have been suggested by some researchers to cause edema and beak malformations, such as crossed beaks, in double-crested cormorants (Firestone 1973; Schrankel et al. 1982; Brunström and Darnerud 1983, all as cited in Brunström 1990).

Mammals

Chronic exposure to PCBs has been shown to cause mortality or serious reproductive complications in mammals. Other effects associated with PCB toxicity include anorexia, liver and kidney degeneration, and gastric ulcers (Wren et al. 1987). Adverse effects on the immune system of marine mammals have also been suggested based on biomarker research (Van Loveren et al. 2000), although the biological significance of the observed biochemical changes is unknown. Like birds, mammals appear to vary widely in their sensitivity to dietary PCBs; reproduction appears to be the most sensitive population-level endpoint for PCB toxicity (Golub et al. 1991; Rice and O'Keefe 1995; Hoffman et al. 1996).

Controlled laboratory exposures of PCBs to mink have been conducted extensively (Aulerich et al. 1985; Wren et al. 1987), and this species appears to be among the most sensitive mammalian species tested (Fuller and Hobson 1986) with reproductive effects as the most sensitive endpoint. A review of the mink toxicity literature indicates that Aroclor 1254 is the most potent Aroclor tested in mink.

In addition, several studies have been conducted with mink that were fed field-collected fish contaminated with a number of organic pollutants, including PCBs, dioxins, furans, and pesticides from Saginaw Bay (Restum et al. 1998). These studies examined the multigenerational reproductive success of captive mink fed these field-collected fish.

Other Organic Compounds

Birds

Relatively few data are available regarding the ecotoxicology of volatile organic compounds and other organic compounds, such as phthalates, to birds. Hexachlorobenzene can be slightly to moderately toxic to birds. The organs affected by hexachlorobenzene exposure are the liver, kidneys, spleen, lungs, and nervous system (EXTOXNET 1996). Phthalates have been suggested as a potential endocrine disruptor for wildlife, although no phthalate studies with birds were found.

Mammals

Data are available for assessing effects of chemicals such as 2-methylphenol, butyl benzyl phthalate, benzidine, and hexachlorobenzene. These chemicals have been

associated with effects ranging from neurotoxicity (2-methylphenol, benzidine) to liver effects, such as alterations in weight (butyl benzyl phthalate, hexachlorobenzene) and increased tumors (hexachlorobenzene) (EPA 1998b).

A.2.4.7.2 Screening methods and results

As previously indicated, screening of wildlife COPCs for this scoping assessment was largely based on the King County Wildlife Risk Assessment (King County 1999), which was a component of the WQA. The King County assessment evaluated ecological and human health risks associated with contaminants in the Duwamish River and Elliott Bay, and the risks resulting from exposure to constituents associated with combined sewer overflows (CSOs). The risk assessment approach and results underwent extensive review by a stakeholder committee and peer review panel, and were accepted by the Washington Department of Ecology. The risk assessment used a probabilistic approach to assess risk to spotted sandpiper, great blue heron, bald eagle, and river otter from ingestion of food and surface water from the area of concern under two scenarios (baseline conditions and without CSOs).

This section describes the county's approach and results under the baseline scenario with CSOs. Included are discussions of their COPC screening, TRV development, exposure assessment assumptions, and risk characterization results. Some of the text in this section contains direct excerpts from King County (1999c) to describe the approach or results. The tables from this document, which are often referred to in this discussion, are attached to this appendix as Attachment A.3.

COPC Screening

King County evaluated 45 candidate COPCs based on analytes included in their 1997 sampling program (Table A-2-15), which included sampling of sediment, surface water, tissue, and CSO discharges. COPCs were selected for further evaluation in the risk assessment using the following screening process. All carcinogens were included while non-carcinogens were screened based on their frequency of detection. For COPCs detected in more than 5% of the water or sediment samples, the 95th percentile water concentration and the 95% UCL on the mean sediment concentration were calculated. If these concentrations were greater than sediment criteria or guidelines or surface water quality criteria, the chemical was included as a COPC. The sediment and surface water quality criteria are presented in Tables 6-1 and 6-2 from Volume III of the WQA (see Attachment A.3).

Table A-2-15. Contaminants of concern evaluated in the King County WQA

METALS/METALLOIDS	ORGANIC COMPOUNDS
Arsenic	1,4-Dichlorobenzene
Cadmium	4-Methylphenol
Copper	Benzo(a)anthracene
Lead	Benzo(a)pyrene
Mercury	Chrysene
Nickel	Benzo(b)fluoranthene
Zinc	Benzo(g,h,i)perylene
(Antimony)	Benzo(k)fluoranthene
(Barium)	BEHP
(Beryllium)	Dibenzo(a,h)anthracene
(Chromium)	Fluoranthene
(Iron)	Indeno(1,2,3-cd)pyrene
(Silver)	Pyrene
(Vanadium)	Phenanthrene
PCBs & PESTICIDES	(2-Methylphenol)
Total PCBs	(Benzidine)
(Aldrin)	(Benzoic acid)
(Aroclor 1254)	(Benzyl alcohol)
(Dieldrin)	(Butyl benzyl phthalate)
(γ-BHC [Lindane])	(Benzo(e)pyrene)
(Heptachlor)	(Dibenzofuran)
ORGANOMETALLICS	(Hexachlorobenzene)
Tributyltin	(Pentachlorophenol)
	(Total HPAH)

Analytes in parentheses were assessed, but were eventually screened out in King County's wildlife risk assessment.

Of the 45 chemicals evaluated, a total of 23 were selected as candidate COPCs for wildlife because they were either known human carcinogens or were frequently detected and exceeded surface water criteria or sediment standards. Of the 22 COPCs not selected, 14 were not detected in any sediment or surface water samples and were excluded. Aroclor 1254 was excluded because it was evaluated as total PCBs. PAH compounds were evaluated on an individual basis, so total HPAH was excluded as a COPC. The remaining infrequently detected COPCs were antimony, barium, benzoic acid, benzidine, iron, and vanadium. These were excluded based on low toxicity or lack of state standards (King County 1999). DDT was not evaluated in the King County WQA. To assess risks to eagle and other avian receptors from DDT, a conservative assessment of risks from DDT to avian receptors was conducted and is presented at the end of this section.

Toxicity Reference Value Development

The King County risk assessment developed TRVs after a review of the following sources:

- U.S. Fish and Wildlife Service contamination review series (e.g., Eisler 1988)
- Agency for Toxic Substance and Disease Registry (ATSDR) (e.g., ATSDR 1991)
- Oak Ridge National Lab database (e.g., Sample et al. 1996)
- General scientific literature

All toxicity studies were evaluated on the relevance of toxic endpoints investigated, and the dosing regime and dosing medium used to expose test organisms. Primary population-level toxicity endpoints included reproduction, development, and survival, while endpoints for assessing risks to individuals (i.e., bald eagle and spotted sandpiper receptors) also included growth reduction and systemic effects such as organ damage. In the absence of toxicological data for the preferred population-level toxicity endpoints for the great blue heron or river otter, effects on growth or other systemic effects were substituted.

In choosing toxicity data to derive TRVs, dosing regimes that most closely represented actual environmental exposures were used when available rather than gavage or intraperitoneal (IP) injection methods. However, data derived using these methods were used if they were the only studies available, as was the case for PAHs.

Due to the general lack of species-specific toxicity studies for the ROCs, data for surrogate species were used. Whenever possible, mammalian toxicity data were used to represent mammalian receptors and avian data to represent avian receptors. However, for 1,4-dichlorobenzene and some PAHs, avian data were not available so mammalian data were used for avian ROCs. To address this additional level of uncertainty, safety factors were applied to these data. A safety factor of 2 was applied for great blue heron to account for interspecies variability, and a safety factor of 5 was applied for the bald eagle and spotted sandpiper to account for interspecies variability and potentially more sensitive endpoints, such as systemic effects or growth. That is, the larger safety factor was applied for the bald eagle and spotted sandpiper to account for risks to individuals as opposed to overall populations. Both species are protected as individuals under the Endangered Species Act (bald eagle) and Migratory Bird Act (spotted sandpiper). These safety factors were based on best professional judgment.

For the river otter, scaling of the toxicity dose was used to adjust the TRVs based on body weight differences between the test species and receptor species. The following formula was used (Sample et al. 1996):

$$NOAEL_w \text{ or } LOAEL_w = NOAEL_t \text{ or } LOAEL_t \left(\frac{BW_t}{BW}\right)^{1/4} \qquad \textit{Equation 2-1}$$

Where:

$NOAEL_w$	=	no observed adverse effects level for mammalian wildlife receptor
$LOAEL_w$	=	lowest observed adverse effects level for mammalian wildlife receptor
$NOAEL_t$	=	no observed adverse effects level for mammalian test species
$LOAEL_t$	=	lowest observed adverse effects level for mammalian test species
BW_w	=	body weight of mammalian wildlife receptor
BW_t	=	body weight of mammalian test species

Body weight scaling was not used for birds because it has not been found to be appropriate (Fischer and Hancock 1997). Instead, a safety factor of 2 for great blue heron and 5 for bald eagle and spotted sandpiper, as described in the above paragraph, were used to account for differences in species sensitivity.

It was assumed for the purposes of the wildlife risk assessment that the toxicity threshold value had an equally likely probability of falling anywhere between the NOAEL and the LOAEL. This probability was represented in the risk characterization by the use of a uniform distribution. When possible, NOAELs were derived from published toxicity literature. When data supporting derivation of a NOAEL were not available for a chemical, the NOAEL was estimated by dividing the LOAEL by a safety factor of 10 (EPA 1996).

The TRVs used for mammals and birds are presented in Tables 2-2 and 2-3 from Appendix B3 of the WQA (see Attachment A.3). The toxicological endpoints for the otter included reproductive effects, such as decreased litter size and reduced fertility, and kidney and liver degeneration. The endpoints for the avian receptors included reproductive effects, such as reduced hatchability and eggshell thinning, kidney damage, and growth reductions. For most chemicals and receptors, reproductive effects were the most sensitive toxicological effect endpoint. For zinc, however, the TRV selected to protect the individual eagle and sandpiper was based on growth effects, which occurred at a lower exposure level than those for reproduction. Toxicity data were available for all chemicals except some PAHs. For these, the toxicological effect data for another PAH, benzo(a)pyrene, were substituted. For the avian receptors, PAH toxicity data were based on mammalian test species for all but fluoranthene and pyrene. For these chemicals, mallard toxicity data were available. To account for the uncertainty of using mammalian toxicity data for avian species, safety factors of 2 to 5 were applied, as previously described.

Exposure Assessment

King County's exposure assessment consisted of characterizing exposure of each of the four wildlife ROCs to COPCs through water, sediment, and food. To characterize exposure of ROCs, several factors were considered including spatial and temporal use of the Duwamish River and Elliott Bay, food and water ingestion rates, incidental sediment ingestion rates, and concentrations of chemicals in exposure media within areas used by ROCs.

Spatial and temporal use

Preferred aquatic habitat of each ROC was defined by King County as a "patch." These patches corresponded to a group of cells in the model grid overlay developed for the Water Quality Assessment study area, which included the Duwamish River and Elliott Bay. Each of these grid cells was further divided into 10 water layers and one sediment layer. The surface water and sediment concentrations from grid cells within each patch for each ROC were used to calculate media ingestion rates of COPCs, as described later in this section. To determine patch areas, site-specific information and the biology of each ROC were considered. Bioavailability of COPCs was assumed to be 100% within each patch.

Two heron patches were defined: one for the period of adult feeding during nesting season (i.e., May - June) and one for the remainder of the year. During the nesting season, the foraging area in the LDW is limited to no more than a mile north or south of the colony near Kellogg Island.[43] During other times of the year the foraging area includes most of the shoreline of the Duwamish River and some areas in Elliott Bay. The patches corresponding to exposure of great blue herons included only the surface layers of the grid cells along most shorelines in the study area.

Bald eagles were assumed to use the entire study area, so the patch included all cells in both Elliott Bay and the Duwamish River. Only the surface layer of water cells was included in the bald eagle patch because bald eagles are unlikely to forage below this water level.

Exposure of spotted sandpipers to COPCs occurs primarily through feeding on invertebrates by picking and probing in intertidal sediments. Spotted sandpiper patches included intertidal shoreline areas in the LDW at Turning Basin 3, on the east side of the river immediately above Slip 4, on Kellogg Island, and on the west side of the river adjacent to Kellogg Island. Several shoreline patch areas were also included in Elliott Bay. Only the surface layer of the water cells was included.

For river otters, the patch area included all shoreline cells at all depths, because of the relatively large home range of the otter.

---

[43] This patch size was based on average distances herons travel between colonies and feeding areas (Mathisen and Richards 1978). It should be noted that this heron colony is no longer an active breeding colony.

Exposure assumptions

The average daily COPC dose received by an ROC was calculated by King County based on food, water, and sediment ingestion rates for a specific ROC. The ingestion rates of various media were in most cases calculated as a function of the ROC body weight. Because risks were evaluated probabilistically,[44] mean body weights and associated standard deviations or standard errors were identified from the literature. Dermal exposure was not evaluated because risks from this pathway are considered to be much less substantial than those from the ingestion pathway (EPA 2000a). In addition, feathers of birds and fur of mammals are believed to limit the contact of the skin surface with contaminated media (EPA 2000a).

Great blue heron

Body weights for both males and females were obtained by King County from EPA's Wildlife Exposure Factors Handbook (1993b), as shown in Table 3-2 from Appendix 3 of the WQA (see Attachment A.3). The food ingestion rate was estimated using an allometric equation (EPA 1993b), as follows:

$$IR_{food} = 10^{0.966 \times \log(BW) - 0.640} \times 0.001\,kg/g \qquad \textit{Equation 2-2}$$

Where:

IR_{food}	=	food ingestion rate (kg/day-wet)
BW	=	body weight (g)

For water ingestion, the following allometric equation was used (EPA 1993b):

$$IR_{water} = 0.059 \times BW^{0.67} \qquad \textit{Equation 2-3}$$

Where:

IR_{water}	=	water ingestion rate (L/day)
BW	=	body weight (kg)

No data on the sediment ingestion rate of great blue heron were found in the literature, however, based on their foraging behavior, it was estimated to be low. Based on best professional judgment, it was assumed that sediment ingestion was equal to 2% of their dietary intake.

Bald eagle

Body weights of both males and females were obtained by King County from the literature (Dunning 1993) and assumed to represent the body weights of eagles in the LDW area. Body weights are shown in Table 3-3 from Appendix B3 of the WQA (see Attachment A.3). Mean values were presented in the literature, but standard

[44] A probabilistic risk assessment is one that uses probabilistic methods to derive a distribution of risk based on multiple sets of values sampled for random variables to incorporate variability and uncertainty into the risk estimate.

deviations on the mean were not available. For use in probabilistic modeling, standard deviations were estimated as one-sixth the range of the body weights. Daily food ingestion rate was assumed to be 12% of body weight (EPA 1993b; Stalmaster 1987). The water ingestion rate was calculated using the same allometric equation as used for great blue heron. Data were not available for sediment ingestion rates, but it was assumed that eagles would ingest some sediment while scavenging along the shoreline. Based on best professional judgment, the sediment ingestion rate was assumed to be 1% of the eagle dietary intake.

Spotted sandpiper

Body weights of both males and females were obtained by King County from the literature (Maxson and Oring 1980), as shown in Table 3-4 from Appendix B3 of the WQA (see Attachment A.3). For use in probabilistic modeling, standard deviations were estimated as one-fourth the range of the body weights. The food ingestion rate was estimated using an allometric equation dependent on body weight (EPA 1993b). The dry weight ingestion rates calculated by this equation were converted to wet weight to ensure conformity with other data used in estimating sandpiper risks. The wet weight ingestion rate was estimated based on 80% moisture in sandpiper food items. The allometric equation used was:

$$IR_{food} = \left(0.0582 \times BW^{0.651}\right) \times \frac{1\,kg\ wet\ matter}{0.2\,kg\ dry\ matter} \qquad \textit{Equation 2-4}$$

Where:

IR_{food} = food ingestion rate (kg/day-wet)
BW = body weight (kg)

The water ingestion rate was estimated using the same equation as for the great blue heron and bald eagle. Sediment ingestion rates for spotted sandpiper were not available, but measured sediment ingestion rates for four other types of sandpiper ranged from 7.3 to 30% (EPA 1993b). The average sediment ingestion rate for the four other sandpipers—18% of diet—was used.

River otter

Body weights for male and female river otter were obtained by King County from the literature (Melquist and Hornocker 1983), as shown in Table 3-5 from Appendix B3 of the WQA (see Attachment A.3). The food ingestion rate was calculated using a model from Iversen (1972) as cited in EPA (1993b), estimated as a function of mean caloric content of prey (k_{prey}), the basal metabolism rate (BMR), and the ratio of free living to BMR (r_{met}). The BMR was calculated as a function of body weight using a uniform probability distribution for body weight. The value of r_{met} was given a uniform probability distribution function, and the value of k_{prey} was given a normal probability distribution function.

For water ingestion rate, an allometric equation from EPA (1993b) was used, as follows:

$$IR_{water} = 0.099 \times BW^{0.90} \qquad \textit{Equation 2-5}$$

Where:

IR_{water}	=	water ingestion rate (L/day)
BW	=	body weight (kg)

Data were not available on the sediment ingestion rate for otters, so a rate equal to 2% of the diet was estimated based on best professional judgment.

Water, sediment, and tissue data used

Exposures for each ROC were estimated by King County based on water and sediment concentrations in their respective patch area(see earlier discussions in this section), as predicted from the water quality model, as well as tissue concentrations from organisms believed to be potential prey species. As part of the WQA, fish and invertebrate tissue samples were collected by King County, in conjunction with the Washington State Department of Fish and Wildlife. The type and number of tissue samples collected are shown in Table 3-16 (in Attachment A.3) from the WQA. Each sample consisted of a composite of organisms to obtain the necessary tissue volume. All tissue samples were collected between Harbor Island and approximately half mile upstream of Kellogg Island, with the exception of one mussel sample collected farther upstream in Slip 4. Amphipod samples were collected just north of Kellogg Island. Samples were analyzed by the King County Environmental Laboratory for organic compounds, including PCBs and PAHs, metals, and butyltins.

For the great blue heron, shiner surfperch data were used to estimate dietary exposure. For the bald eagle, shiner surfperch and salmon data were used, with the assumption that perch and combined salmon species were consumed in equal proportions. Amphipod data were used for spotted sandpiper exposure. For river otter exposure estimates, shiner surfperch, mussels, crab hepatopancreas, and crab meat data were used, with the assumption that each prey type was consumed in equal proportions.

The estimated exposure concentrations (EECs) for water and sediment are shown in Tables 3-6 through 3-10 from Appendix B of the WQA (see Attachment A.3). Note that data are presented in these tables for both scenarios (with and without CSOs), although this appendix is focusing only on results from current baseline conditions *with* CSOs. The tissue EECs for wildlife receptor prey items are presented in Table 3-11 (see Attachment A.3). Depending upon the receptor, data for water, sediment, and tissue may represent samples collected in both Elliott Bay and the LDW. Uncertainties associated with these exposure results compared to exposure from only the LDW are discussed in the uncertainty assessment (Section A.7.3.2).

Methods

The estimated environmental dose (EED) of each contaminant was calculated separately for water, food, and sediment for each ROC, using the following equation:

$$EED_{media} = EEC_{media} \times \left(\frac{0.5 \times MIR_m}{BW_m} + \frac{0.5 \times MIR_f}{BW_f} \right) \qquad \textit{Equation 2-6}$$

Where:

EED_{media}	=	estimated environmental dose in media (water, food, or sediment) (mg/kg BW/day)
EEC_{media}	=	estimated chemical concentration in media (mg/kg or mg/L)
MIR	=	media ingestion rate (kg/day or L/day)
BW	=	body weight of receptor (kg)
m	=	male
f	=	female

As shown in the equation, EEDs were calculated assuming the receptor populations were 50% male and 50% female.

The total dose to each ROC was estimated by combining the water, food, and sediment EEDs, as shown in the following equation:

$$EED_{total} = EED_{sed} + EED_{water} + EED_{food} \qquad \textit{Equation 2-7}$$

Where:

EED_{total}	=	total expected environmental dose to ROC (mg/kg BW/day)
EED_{sed}	=	sediment dose to ROC (mg/kg BW/day)
EED_{water}	=	water dose to ROC (mg/kg BW/day)
EED_{food}	=	food dose to ROC (mg/kg BW/day)

The EEDs were not adjusted using bioavailability factors or site use factors. Instead, it was conservatively assumed that the COPCs in media were 100% bioavailable, and that each ROC forages entirely within the LDW.

Risk Characterization

Risks to each ROC from the selected COPCs were estimated using the hazard quotient (HQ) approach, where:

$$\text{Hazard Quotient} = \frac{\text{Expected Environmental Dose}}{\text{TRV}} \qquad \textit{Equation 2-8}$$

HQs were determined for each media exposure pathway separately, and then summed to determine the HQ for all exposure pathways combined. HQs were determined by exposure pathway to identify which pathway contributed most to the total risk for each species.

Probabilistic methods were used to derive distributions of HQs. Monte Carlo analysis was used to repeatedly perform the HQ calculation using randomly selected sets of input values each time. Variables in the model included ROC body weights and dietary tissue concentrations, which were based on a normal distribution using the mean and standard error. Food, sediment, and water ingestion rates also varied because they were a function of body weight. Chronic toxicity thresholds varied assuming a uniform distribution between the NOAEL and LOAEL. The only parameters that did not vary were the mean surface water and sediment concentrations derived from the model. The results were summarized based on the mean and the 5th and 95th percentile of the distribution. The 5th and 95th percentile HQs were used to represent the lower and upper bounds of the probability of risk.

For great blue heron and bald eagle, none of the mean HQs for any COPC exceeded 1 (see Attachment A.3; Tables 4-16 and 4-19 from Volume 1 of the WQA). The only 95th percentile HQ to exceed 1 was for lead, which had an HQ of 1.8 for heron and 2.0 for eagle. The sediment ingestion pathway contributed the greatest risk for both ROCs. For PCBs, neither the mean nor 95th percentile HQs exceeded 1 for great blue heron or bald eagle.

For the spotted sandpiper, mean HQs exceeded 1 for copper, lead, zinc, PCBs, and bis(2-ethylhexyl)phthalate (BEHP) (see Attachment A.3; Table 4-10 from Volume 1 of the WQA). The 5th percentile, mean, and 95th percentile HQs were 16, 22, and 27 for copper; 46, 112, and 279 for lead; 0.5, 1.4, and 2.4 for zinc; 1.5, 2.5, and 3.7 for PCBs; and 0.4, 2.3, and 4.2 for BEHP. These HQs are driven primarily by the ingestion of prey (i.e., amphipods).

For the river otter, the only COPC with a mean HQ exceeding 1 was lead (mean HQ = 1.6; see Attachment A.3; Table 4-13 from Volume 1 of the WQA). Lead and arsenic were the only COPCs with 95th percentile HQs exceeding 1 (3.8 for lead and 1.1 for arsenic). These HQs are driven by the ingestion of prey (i.e., crabs, mussels, and shiner surfperch).

In summary, the ROC/COPC pairs in Table A-2-16 had 95th percentile HQs greater than 1, indicating a greater potential for risk. These pairs will be further evaluated in this Phase 1 ERA for the LDW, with emphasis on sediment-related pathways and exposure data, and a thorough assessment of the available effects data (Sections A.5.1 and A.5.2).

Table A-2-16. ROC/COPC pairs with 95th percentile HQs greater than 1 (based on King County [1999])

ROC	METALS	ORGANICS
Great blue heron	Lead	
Bald eagle	Lead	
Spotted sandpiper	Copper, lead, zinc	PCBs, BEHP
River otter	Lead, arsenic	
Harbor seals[a]		

[a] Harbor seals were not evaluated in King County (1999c).

Additional screens

In addition to the ROC/COPC pairs listed in Table A-2-16, PCBs were added as a COPC for both great blue heron and bald eagle for evaluation in the exposure and effects assessment. PCBs were added for great blue heron because recent PCB egg concentration data collected from the West Seattle colony were elevated compared to TRVs (Section A.7.3). PCBs were added for eagle at the request of the agencies.

An additional conservative screen was conducted for risk to piscivorous avian ROCs[45] from DDT and mercury using similar calculations as the King County assessment but without a probabilistic approach. Instead, the approach presented in Section A.5.1.1 of this document was used to calculate exposure doses for great blue heron and eagle. As a conservative approach, maximum concentrations of DDT and mercury measured in fish and sediment were used. The exposure factor values used were the same as those presented in Table A-5-6, and it was assumed that both heron and eagle obtained all their food from the LDW.

The avian DDT TRV used was a NOAEL of 0.084 mg/kg bw/day derived from Lincer (1975). Lincer (1975) exposed American kestrels in the laboratory to DDE and noted significant thinning of eggshells at dietary doses of 3.0 mg/kg and higher, but not at 0.3 mg/kg. The assumed body weight was 0.114 kg (California EPA 2002) and the calculated food ingestion rate was 32 g/day (wet weight basis; calculated from Nagy 1987).[46] This study presented the lowest laboratory avian effect concentration found for a DDT or its metabolites from database searches (including Cal/EPA, ECOTOX, and BIOSIS), and a search of the scientific literature. Although a lower LOAEL of 0.028 mg/kg/day was presented in Sample et al. (1996), this value was from a study that observed reproductive success of wild brown pelicans in the field. It was not considered appropriate for this evaluation because of the lack of controls and potential for multiple chemical exposures. The fish and sediment exposure concentrations, calculated exposure doses, NOAELs, and HQs are presented in Table A-2-17. The resulting NOAEL-based HQs for great blue heron and eagle were 0.13 and 0.08,

[45] Data were not available for DDT in amphipod tissue to screen for risk to sandpiper from DDT exposure.

[46] Dry weight ingestion rate calculated from Nagy was converted to wet weight assuming 33% solids in food.

respectively. Based on the screening, DDTs were not selected for further evaluation for birds in the Phase 1 ERA. However, due to the lack of data for sandpiper and the potential for DDT to biomagnify, the feasibility and utility of collecting additional exposure data for all DDT/bird pairs will be further discussed in the data gaps process.

For mercury, exposure data and HQs are presented in Table A-2-17. NOAEL-based HQs for both great blue heron and eagle exceeded 1 (2.1 and 1.3, respectively) based on a NOAEL of 0.0091 mg/kg bw/day from a study with great egrets (Spalding et al. 2000; derivation of this TRV is discussed in Section A.5.2.1.4). As such, mercury was retained as a COPC for heron and eagle[47] and further evaluated in Section A.5.

Table A-2-17. DDT and mercury data and HQs for additional screen of risk to heron, eagle, and seal

	Maximum Fish Conc.[a] (mg/kg dw)	Maximum Sediment Conc. (mg/kg dw)	Exposure Dose[b] (mg/kg bw/day)	NOAEL (mg/kg bw/day)	NOAEL HQ
Heron					
DDT	0.204	2.88	0.011	0.084[c]	0.13
Mercury	0.367	4.6	0.019	0.0091[d]	**2.1**
Eagle					
DDT	0.204	2.88	0.0067	0.084[c]	0.08
Mercury	0.367	4.6	0.012	0.0091[d]	**1.3**
Seal					
DDT	0.204	2.88	0.002	0.80[e]	0.003
Mercury	0.367	4.6	0.0026	0.017[f]	0.20

Note: HQs greater than 1.0 are noted in bold type.

[a] Highest concentration of mercury was measured in shiner perch. Highest concentration of DDT was measured in juvenile chinook salmon, which were the only fish tissue samples analyzed on a whole body basis. Wet weight values were converted to dry weight using average moisture content in English sole (76%).

[b] The food ingestion rate, sediment consumption rate, and body weight values used to calculate exposure doses for heron, eagle, and seal are presented in Table A-5-8.

[c] Derived using a no-effect dietary concentration of 0.3 mg/kg ww (Lincer 1975), a body weight of 0.114 kg (California EPA 2002), and a food ingestion rate of 0.032 kg ww/day (calculated from Nagy 1987).

[d] Derived using a no-effect dietary concentration of 0.05 mg/kg ww (Spalding 2000), a body weight of 1.02 kg (Arizona Game and Fish 2002), and a food ingestion rate of 0.185 kg ww/day (Kushlan 1978).

[e] Derived using a no-effect dietary concentration of 10 mg/kg ww (Fitzhugh 1948), a body weight of 0.35 kg (Sample et al. 1996), and a food ingestion rate of 0.028 kg ww/day (Sample et al. 1996).

[f] Derived using a no-effect dietary concentration of 0.017 mg/kg ww (Wobeser et al. 1976), a body weight of 0.975 kg (Aulerich and Ringer 1977), and a food ingestion rate of 0.150 kg ww/day (Bleavins et al. 1980).

[47] Risk to sandpiper from mercury was found to be low in the King County Wildlife Risk Assessment (1999c), so this pair was not evaluated further.

For mammals, recent studies of wild populations in the Baltic Sea have suggested a link between mass mortalities in harbor seals and exposure to chlorinated organic contaminants, including pesticides, PCBs, and PCDDs/PCDFs, leading some researchers to suggest an immunotoxicological effect associated with this class of compounds (Ross et al. 1996). These potential immunotoxicological effects are discussed in Section A.5, and PCBs were added as an ROC/COPC pair for potential effects to seal reproduction, growth, and mortality.

To assess risks to seals from mercury and DDT exposure, a conservative screen was conducted using maximum fish tissue concentrations and assuming seal feed entirely on fish from the LDW (i.e., site usage factor (SUF) of 1). The mercury TRV was derived from a study by Wobeser et al. (1976) exposing mink for 93 days, which identified a NOAEL for mortality, weight loss, and ataxia at a methylmercury dose of 0.17 mg/kg bw/day. This study was considered subchronic according to guidelines from Sample (1996), which selected one year as the minimum exposure period to represent chronic exposure for mammals. Because this study was neither chronic nor conducted during a sensitive lifestage, such as reproduction, an uncertainty factor of 10 was used to derive a NOAEL of 0.017 mg/kg bw/day, as recommended by Sample (1996). Exposure data and HQs are presented in Table A-2-17. The DDT TRV for seals of 0.80 mg/kg bw/day was derived from a study of effects in rats by Fitzhugh (1948). In this study, rats experienced reproductive effects when exposed to DDT for 2 years at a dietary concentration of 50 mg/kg, but not at 10 mg/kg. HQs for mercury and DDT based on NOAELs were less than 1 for seal (0.20 and 0.003, respectively). Based on this assessment, mercury and DDT were not retained for further evaluation in the effects and exposure assessment for seals.

An additional preliminary evaluation of PCB risk to river otter based on a conservative, worst-case exposure scenario (i.e., entire diet consisting of fish with maximum detected PCB concentration) indicated potential risks to reproduction are possible for otter. Therefore, the river otter/PCBs pair was retained for further evaluation in the ERA (Section A.5).

In summary, in addition to the COPCs retained based on the King County wildlife assessment (Table A-2-16), PCBs were retained as a COPC for all wildlife species, and mercury was retained as a COPC for great blue heron and bald eagle. These ROC/COPC pairs are further analyzed in the effects and exposure assessments (Sections A.5.1 and A.5.2) and risk characterization (Section A.7.3). The potential to collect additional data to assess the DDT/bird pairs will be further discussed in the data gaps process.

A.2.4.8 Plants

This section presents a brief summary of ecotoxicological data for plants, as well as the methods and results of the COPC screen for aquatic rooted plants.

A.2.4.8.1 Ecotoxicology

Metals

Metals are natural elements and are ubiquitous in trace concentrations in the different compartments of the aquatic ecosystem; some are essential micronutrients for plants. Plants easily accumulate zinc, copper, cadmium, nickel, and other metals. General physiological processes (e.g., transpiration, respiration, and photosynthesis) and plant development in general can be inhibited by elevated exposure to some metals (Wallnofer and Engelhardt 1984, as cited in Vangronsveld and Clijsters 1994). Stunted growth, leaf epinasty, and chlorosis are symptoms of metal toxicity. Lesser effects include plasma membrane permeability leading to leakage of ions and other solutes, and inhibition and stimulation of enzymes (Vangronsveld and Clijsters 1994).

The uptake of mercury in plants has been studied extensively, and mechanisms leading to reduced growth and survival are documented. For example, it has been shown that excess amounts of Hg^{2+} result in lower rates of oxygen consumption in aquatic plants, leading to inhibited growth (Jana and Choudhuri 1982). Mercury may affect membrane transport proteins or metabolic events that in turn affect the osmotic relations of root tissues and water transport, ultimately inhibiting the hydraulic conductivity in roots (Maurel 1997).

No data were found on the toxicity of TBT to plants.

Pesticides

Pesticides represent a large group of compounds designed to kill, repel, or regulate the growth of undesirable organisms. Pesticides include fungicides, herbicides, nematicides, molluscicides, rodenticides, fumigants, disinfectants, repellents, wood preservatives, and antifoulants (PSWQA 1990). Herbicides affect plants by several different modes of action including inhibition of photosynthesis, inhibition of protein synthesis, inhibition of amino acid biosynthesis, disruption of plant cell membranes, light-activated free radical formation, disruption of respiration, disruption of cell division and general growth inhibition (Fleming et al. 1995).

PAHs

PAHs are hydrophobic and readily partition from air and water into the cuticular layers and lipoprotein membranes of plants (Seuss 1976; Southworth et al. 1978; Cook et al. 1983). PAHs are metabolized by plants and the breakdown products can be more toxic and mutagenic than the parent compounds. For example, PAHs are oxidized by cytochrome p-450 to more potent toxicants. PAHs strongly absorb UV radiation (200-400 nm) and are prone to light-induced chemical modification (Huang et al. 1991). UV radiation has been found to significantly enhance the toxicity of PAHs to plants.

PCBs

Little information is available relating PCBs to plant toxicity. Effects of PCBs on algae reported in the EPA AQUIRE database include mortality, reduced growth, reduced photosynthesis, and reduced chlorophyll content.

Other Organic Chemicals

Elevated exposure to di-n-butyl phthalate can result in reduced growth and a reduction in seed germination (Overcash et al. 1982, as cited in Efroymson et al. 1997). Elevated exposure to pentachlorophenol can result in reduced fresh weight of shoots (Gunther and Pestemer 1990, as cited in Efroymson et al. 1997). Phenols and anilines can result in inhibition in the rate of root elongation (Feng et al. 1996, as cited in Efroymson et al. 1997).

A.2.4.8.2 Screening methods and results

Plants were screened for potential adverse effects of COPCs by comparing the maximum concentrations measured in intertidal sediment in the LDW to screening benchmarks presented in Efroymson et al. (1997) that were developed to assess the hazard to terrestrial plants caused by contaminants in soil based on either field tests or studies in greenhouses (more common). Soil benchmarks were compared to chemical concentrations in LDW sediments to provide an indication of risks because toxicity data for effects to emergent aquatic plants exposed to contaminated sediments are not available. The use of soil toxicity benchmarks as a surrogate for sediment evaluation is discussed in the uncertainty assessment (Section A.7.4.2).

In addition to a comparison to these benchmarks, chemical concentrations in intertidal and marsh sediments were also compared to background concentrations in sediments and soils, as suggested by Efroymson et al. (1997). According to Efroymson et al. (1997), if vigorous and diverse plant communities are present at sites with concentrations greater than these benchmarks, or if background concentrations at a site exceed these benchmarks, then these benchmarks can be considered poor indicators of risks to plants at a given site. Variations in responses of plant communities to chemicals in soil indicate that there is a wide range of sensitivity of different plants to chemicals and that site-specific soil conditions affect chemical availability. Background concentrations used in this screen are based on background concentrations in the Puget Sound area from the Puget Sound Estuary Program (Tetra Tech 1988), Washington Department of Ecology's Toxic Cleanup Program (for soils) (Ecology 1994), and reference area performance standards for Puget Sound sediments (PTI 1991).

All chemicals for which Efroymson et al. (1997) benchmarks were available were screened; in total, 14 benchmarks are presented for chemicals for which LDW sediment data are available (Table A-2-18). Acenaphthene, diethylphthalate, di-n-butyl phthalate, pentachlorophenol, and phenol had maximum concentrations in intertidal

sediments less than the toxicity-based benchmarks from Efroymson et al. (1997), and thus were assumed to pose negligible risks to plants.

Concentrations of the remaining COPCs in intertidal and marsh sediments were compared to background concentrations in sediments and soils (Table A-2-19). All intertidal sediment data were included in this screen because emergent aquatic plants could potentially grow in the upper zones of this area. However, plants do not necessarily grow throughout the entire intertidal area because various other conditions preclude their growth (primarily salinity and percent tidal immersion), limiting the growth of plants to a few marsh areas. Cordell et al. (1999) makes reference to a number of conditions, including riprapping, wave action, prop wash, goose grazing, and industrial and woody debris, that are likely responsible for the current distribution of plants, within their normal salinity and tide zones. Thus, marsh data were viewed as more representative of plant exposure. Note, however, that far fewer data were available for marsh areas than intertidal areas (see Section A.7.4.2 for a discussion of uncertainty).

Table A-2-18. Plant COPC screen using terrestrial plant screening values from Efroymson et al. 1997

Chemical	Maximum Intertidal Sediment Concentration (mg/kg)	Benchmark concentration (mg/kg)
Acenaphthene	0.76	20
Arsenic	79	10
Cadmium	92	4
Chromium	1,100	1
Copper	12,000	100
Diethylphthalate	0.26	100
Di-n-butyl phthalate	3.8	200
Lead	23,000	50
Mercury	4.6	0.3
PCBs (total-calculated)	223	40
Pentachlorophenol	1.9	3
Phenol	2.1	70
Silver	270	2
Zinc	6,400	50

Table A-2-19. Comparison of sediment COPC concentrations in marsh and intertidal habitat to Puget Sound background concentrations

Chemical	Range (and 95% UCL on the mean)[a] Sediment Concentration in Intertidal (mg/kg dw)	Range (and 95% UCL on the mean)[a] Sediment Concentration in Marshes (mg/kg dw)[b]	Background Soil and Sediment Concentration Range (mg/kg dw)
Arsenic	1.9 – 79 (12)	6.4 – 17 (14))	1.8-17[c]; 17[d]; 22[e]
Cadmium	0.020 – 92 (2.7)	0.10 – 0.60 (0.50)	0.047-1.9[c]; 5[d]; 1.5[e]
Chromium	5.0 – 1,100 (63)	15 – 35 (33)	9.6-255J[c]; 235[d]; 85[e]
Copper	5.0 – 12,000 (240)	22 – 90 (72)	5-74[c]; 243.5[d]; 53[e]
Lead	2.0 – 23,000 (410)	9.3 – 330 (160)	0.1U-24[c]; 29.6[d]; 20[e]
Mercury	0.020 – 4.6 (0.24)	0.090 – 0.37 (0.25)	0.01-0.28[c]; 0.0944[d]; 0.15[e]
PCBs (total-calculated)	0.00030 – 223 (3.0)	0.020 – 9.4 (1.7)	0.0031-0.050U[c]; 0.047[e]
Silver	0.060 – 270 (3.9)	0.070 – 0.53 (0.44)	0.02U-3.3[c]; 0.32[e]
Zinc	16 – 6,400 (312)	56 – 160 (133)	15-101J[c]; 132.5[d]; 103[e]

U - Undetected

J - Estimated

a Nondetects were treated as half the detection limit in the 95% UCL calculations

b Maximum concentrations of COPC within 50 m of marsh habitat (per USFWS designation) (n=7 stations: DR013, DR014, DR061, DR263, DR264, DR270, DR271; see RI Maps 2-5a through 2-5k)

c PTI (1991) (range of concentrations from Puget Sound sediment reference areas)

d Ecology (1994) (maximum concentration in Puget Sound soil reference areas)

e PTI (1991) (proposed reference area performance standard [i.e., sites with concentrations lower than these standards are suitable for reference area classification])

Based on a comparison of sediment COPC concentrations in marsh and intertidal areas to background concentrations (Table A-2-19), COPCs were placed in two groups. The first group—arsenic, cadmium, chromium, copper, and silver—have maximum concentrations in marsh areas that fell within the range of background concentrations. In addition, the 95% UCL of the mean concentrations in intertidal sediment of three of these five chemicals (exceptions were cadmium and silver) were within the range of background concentrations. Thus, elevated exposure of these contaminants to plants is unlikely to be above background levels, and these COPCs were assumed to pose negligible risk and are not evaluated further in the Phase 1 ERA (Table A-2-20).

The second group—lead, mercury, zinc, and PCBs—have 95% UCL mean and maximum concentrations in marsh areas that were greater than background concentrations (except for 95% UCL of the mean concentration of mercury and zinc in marsh areas, which were similar to background concentrations). Exposure and effect concentrations for these COPCs are discussed further in Sections A.6 and A.7.4.

Table A-2-20. Comparison of sediment chemical concentrations in LDW intertidal and marsh areas relative to background concentrations.

Chemical	Intertidal vs. Background Concentration	Marsh vs. Background Concentration	Retain for further Assessment in Phase 1 ERA?
Arsenic	95% UCL within range of sediment background; max > sediment/soil background	max and 95% UCL within background sediment range	no
Cadmium	95% UCL within range of sediment/soil background; max > sediment/soil background	max and 95% UCL within background sediment range	no
Chromium	95% UCL and max > sediment/soil background	max and 95% UCL within background sediment range	no
Copper	95% UCL within range of soil background; max > sediment/soil background	95% UCL within background sediment range; max < soil background	no
Lead	95% UCL and max > sediment/soil background	95% UCL and max > sediment/soil background	yes
Mercury	95% UCL similar to sediment background; max > sediment/soil background	95% UCL within the background sediment range; max > sediment/soil background	yes
PCBs	95% UCL and max > sediment/soil background	95% UCL and max > sediment/soil background	yes
Silver	95% UCL and max > sediment background	max and 95% UCL within background sediment range	no
Zinc	95% UCL and max > sediment/soil background	95% UCL and max > sediment/soil background	yes

A.2.5 Site Conceptual Model

This section presents the site conceptual model that synthesizes the dynamics of the LDW[48] and how ROCs may be exposed to chemical stressors. Based on the model presented here, assessment endpoints and measures of exposure and effect are selected and discussed. These assessment endpoints determine which endpoints will be examined in detail in this Phase 1 ERA for each ROC/COPC combination that was retained for further analysis based on the analyses in Section A.2.4.

A.2.5.1 Exposure pathways

This section discusses the potential for ROCs in the LDW to be exposed significantly to COPCs. For COPCs to pose risk to ROCs, the exposure pathway must be complete. Identifying complete exposure pathways prior to a quantitative evaluation allows the assessment to focus on only those chemicals that can reach ecological receptors (EPA 1997a,b). An exposure pathway is considered complete if a chemical can travel from a source to ecological receptors and is available to the receptors via one or more

[48] The primary sources of chemicals to the waterway are not addressed here. Available source information is discussed in the Phase 1 RI (Section 4.3).

exposure routes (EPA 1997a,b). Complete pathways can be of varying importance, so it is important to identify key pathways that reflect maximum exposures to ecological receptors sensitive to that chemical (EPA 1997a,b).

Exposure pathways for sediment-associated chemicals to ROCs in the LDW were designated in one of four ways: complete and significant, complete and significance unknown, complete and insignificant, or incomplete. Each of the four designations is defined below to provide a clear explanation of how it will be addressed in the ERA process. This section also presents a brief rationale for each designation by receptor. The conceptual site model is presented in Figures A-2-2 and A-2-3 for aquatic species and wildlife, respectively.

- *Complete and significant:* There is a direct link between the receptor and pathway, and the specific pathway is considered a potentially important driver for risk. Pathways classified as "complete and significant" will be addressed in greater detail in the exposure and effects assessment (Sections A-3, A-4, A-5, and A-6).
- *Complete and significance unknown:* There is a direct link between the receptor and the pathway; however, there is insufficient scientific data available to quantify the significance of the pathway in the overall assessment of exposure. Pathways classified as "complete and significance unknown" will be discussed qualitatively in the uncertainty assessment (Section A.7).
- *Complete and insignificant:* There is a direct link between the receptor and the pathway: however, the significance of this pathway in terms of overall exposure is considered to be negligible. Pathways classified as "complete and insignificant" will not be evaluated further in the Phase 1 ERA.
- *Incomplete:* There is no direct link between the receptor and the pathway. Pathways classified as "incomplete" will not be evaluated further in the Phase 1 ERA.

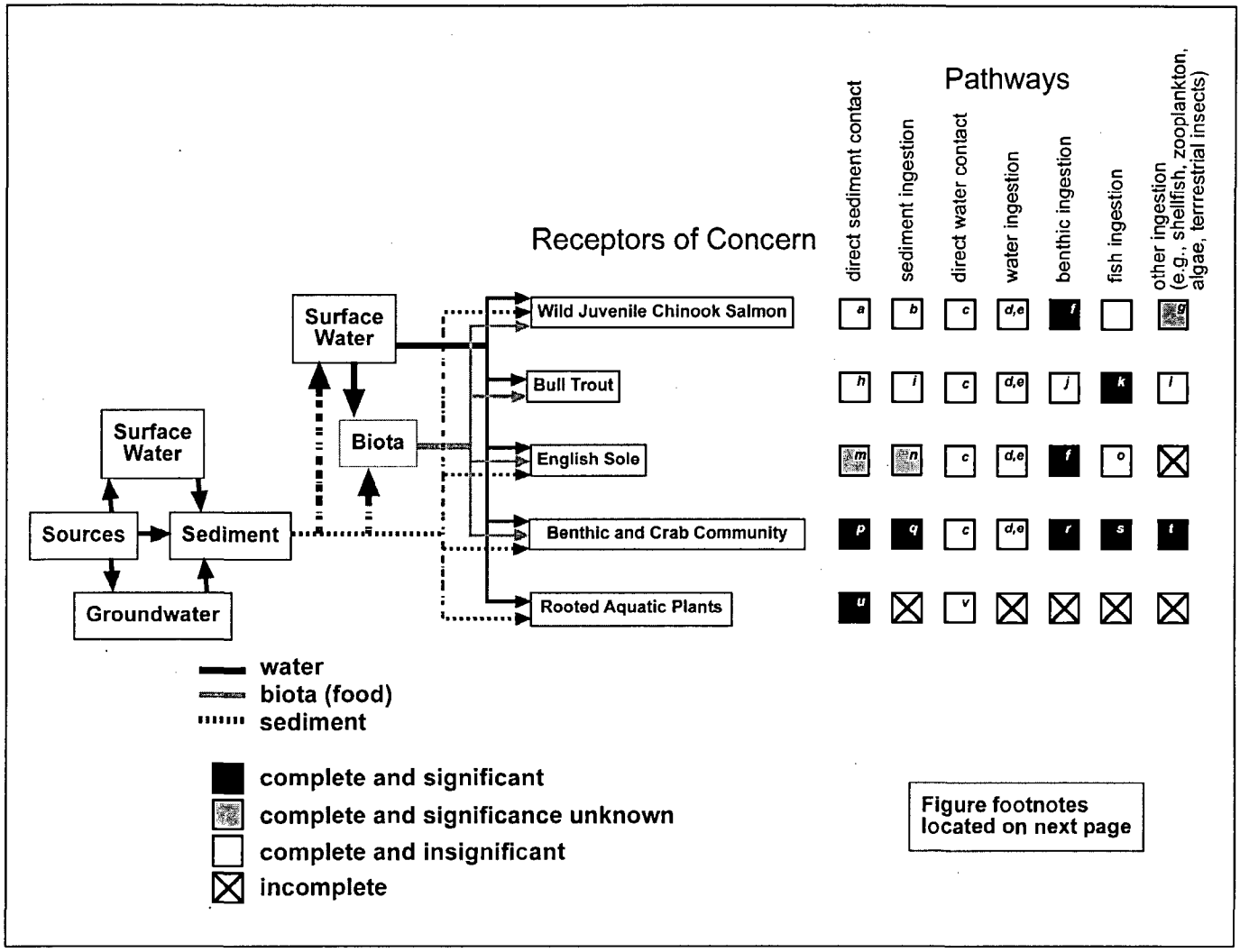

Figure A-2-2. Conceptual site model for fish, invertebrate benthic community, and plants

Notes for Figure A-2-2

a Juvenile chinook do not come into direct contact with sediment for a significant period of time; therefore, any exposure via direct sediment contact is considered insignificant in the overall exposure assessment.

b Because juvenile chinook are generally not in direct contact with sediment, this exposure pathway is likely insignificant. Examination of juvenile chinook stomach content suggests they do not ingest an appreciable amount of sediment (Cordell 2001b).

c Aquatic organisms are in direct contact with surface waters; based on a comparison of modeled concentrations of contaminants in water to ambient water quality criteria (King County 1999), risks to aquatic life through the water pathway appear to be low (Attachment A.2).

d Based on a comparison of modeled concentrations of contaminants in water to ambient water quality criteria (King County 1999c), risks to aquatic life appear to be low (Attachment A.2).

e Aquatic organisms may ingest water; however, the significance of this exposure pathway for sediment-associated chemicals is unknown.

f Epibenthic invertebrates are assumed to be a primary component of the diet.

g Juvenile chinook may occasionally consume drift organisms; however, the contribution of this component to the overall diet is unknown.

h In general, bull trout do not come into direct contact with sediment. Therefore, any exposure via direct contact with sediment is considered insignificant in the overall exposure assessment.

i Because bull trout are generally not in direct contact with sediment, this exposure pathway is likely insignificant. Examination of bull trout prey (i.e., chinook salmon) stomach content suggests they do not ingest an appreciable amount of sediment (Cordell 2001b).

j Ingestion of benthic invertebrates is assumed to be a very small component of the overall bull trout diet. Also, worst-case exposure results in low risk (see Section A.7.2.2).

k Fish are considered a primary component of the bull trout diet.

l Bull trout are opportunistic feeders and may occasionally consume other prey items (e.g., water column invertebrates, drift organisms). The overall contribution of this component to the bull trout diet, however, is assumed to be insignificant.

m Sole routinely bury themselves in sediment, and so are in direct contact with sediment and associated porewater. However, no data are available to estimate risk from direct contact.

n Sole reside and forage in the sediments and they are likely to consume some sediment and associated porewater; however, the specific amount consumed is unknown. It is assumed to be 10% in Section A.4.1.2 based on best professional judgment.

o English sole are not known to ingest fish (Hart 1973). Therefore, the significance of this exposure pathway for sediment-associated chemicals is considered insignificant.

p Benthic organisms are generally in direct contact with sediment and associated porewater.

q Some benthic organisms are known to routinely ingest sediment, and therefore, this pathway is considered complete and significant.

r A significant portion of the diets of some benthic organisms consists of other benthic organisms.

s Although benthic organisms generally do not ingest fish, crabs may ingest dead fish.

t Benthic organisms may ingest algae and detritus.

u Rooted plants are in direct contact with sediment and associated porewater.

v Rooted plants are rarely submerged in the LDW.

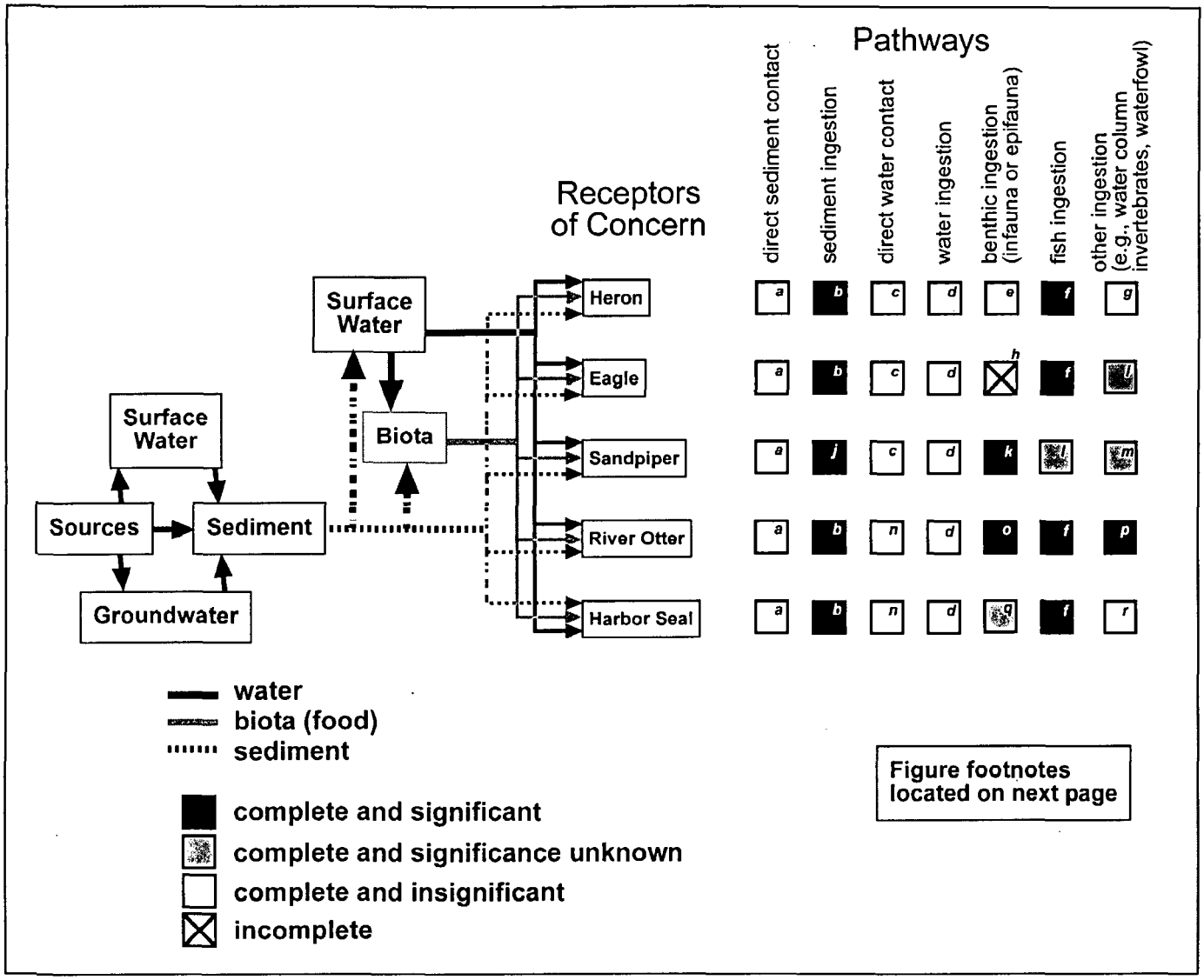

Figure A-2-3. Conceptual site model for wildlife

Notes for Figure A-2-3

a Species may come in contact with sediment when foraging; however, no data are available to assess risks through this pathway. It is generally considered insignificant.

b Species may incidentally ingest a small amount of sediment while foraging.

c Species come in contact with surface water when foraging. Although no data are available to assess risks through this pathway, risks to wildlife through this pathway are generally assumed to be insignificant.

d Based on King County (1999c), risk from water ingestion accounted for less than 0.5% of the overall risks.

e Great blue heron may occasionally consume benthic organisms, but benthic organisms make up a very small component of their diet (EPA 1993b).

f Fish are the primary component of the diet.

g Great blue heron may consume aquatic insects, but insects are not reported to represent a high percentage of their diet (EPA 1993b). In addition, herons may also consume amphibians; however, amphibians have not been observed in the LDW, with the exception of a single tadpole.

h Bald eagles generally do not consume benthic invertebrates.

i Bald eagles may consume birds, such as grebes, gulls and waterfowl, and may infrequently consume mussels (EPA 1993b). However, no data are available on body burdens in birds or the percent of those body burdens that could be attributable to sediment sources. Eagles may also infrequently feed on marine mammal carcasses, but this pathway is considered insignificant at this site.

j Sandpipers can ingest sediment (assumed to be 18% of diet [EPA 1993b]) when foraging or from their food.

k Benthic organisms are a primary component of the sandpiper's diet.

l Spotted sandpipers may occasionally consume small fish, but the percentage of fish in the overall diet and their significance is unknown.

m Spotted sandpipers ingest terrestrial insects (Terres 1987) and may ingest mollusks (e.g., mussels). For terrestrial insects, however, there is no direct connection attributable to sediment sources. The percent mollusk ingestion in the diet and relative importance of sediment-contaminants to mollusk body burdens are unknown, and therefore, the significance of this diet component is unknown.

n Species are in direct contact with surface water when swimming and foraging. Although no data are available to assess risks through this pathway, risks to wildlife through this pathway are generally assumed to be insignificant.

o River otters may ingest crabs as a significant proportion of their diet (Larsen 1984; Stenson et al. 1984; EPA 1993b).

p Mussels may make up a significant portion of the otter's diet.

q Harbor seals may consume crabs as a part of their diet, but the relative percentage is unknown.

r Although squid and octopus can be important prey items, they have not been observed in the LDW, and are thus unlikely to make up a significant portion of the seal diet in the LDW.

A.2.5.2 Food-web model

To understand the potential exposure pathway of a sediment-associated chemical to upper trophic level ROCs, knowledge of food-web relationships is important. Figure A-2-4 depicts a generalized food web for the LDW. The food web shows the relationship between major trophic groups, and lists several representative species.[49] The relationship among trophic groups illustrates the potential pathways for chemical transfer throughout the LDW food web. Thus, Figure A-2-4 provides additional detail for the prey ingestion pathways identified in Figures A-2-2 and A-2-3.

[49] Note that some organisms could have representatives in more than one box, depending on their diversity and life stage.

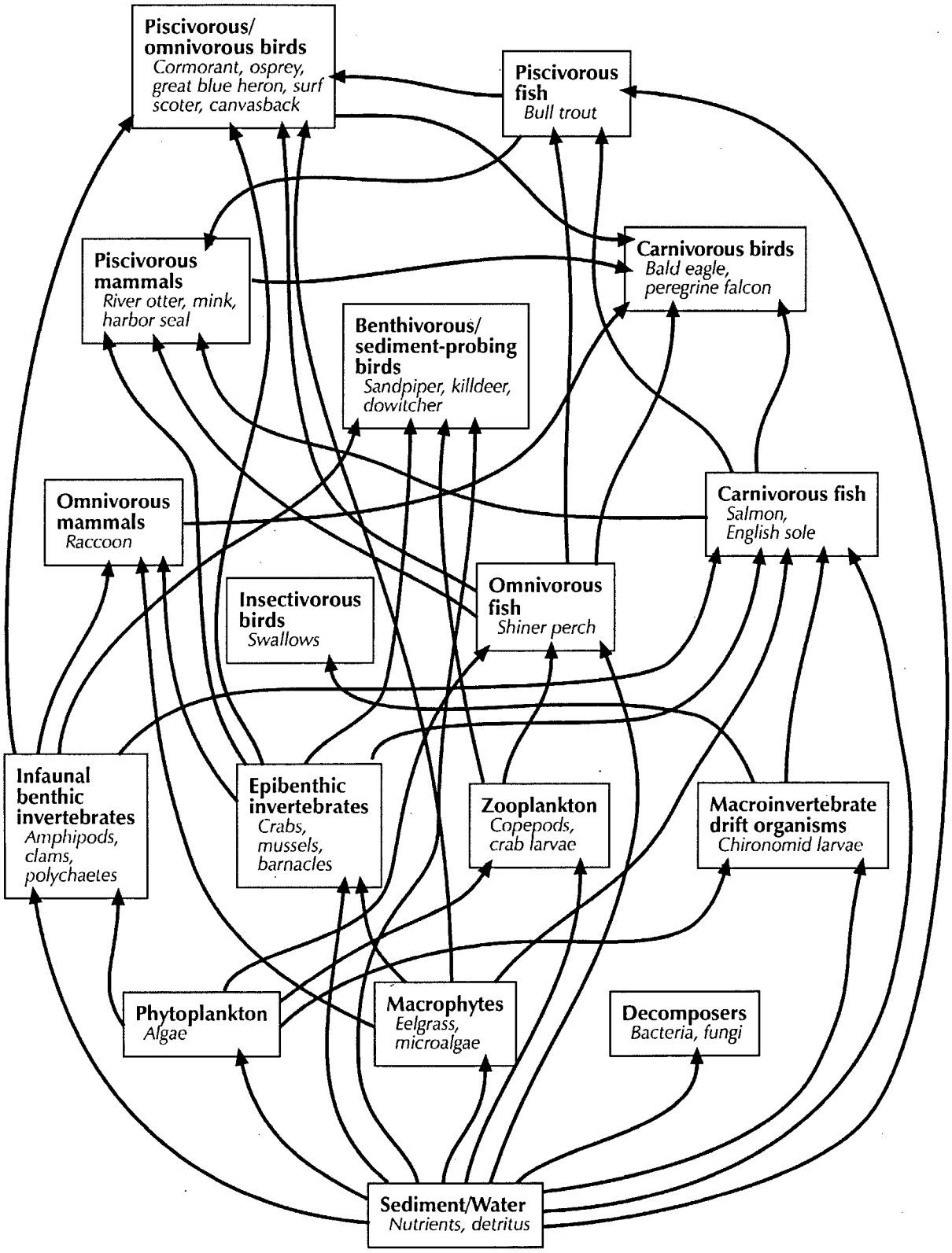

Figure A-2-4. Generalized food-web model for the LDW

A.2.5.3 Summary of ROC/COPC pairs and pathways

In summary, benthic invertebrates (including crabs) are primarily exposed to COPCs in sediment through ingestion of sediment or porewater, direct contact with sediment, and through feeding on contaminated prey (Figure A-2-2). COPCs for benthic species include TBT, all of the SMS chemicals, and 15 of the DMMP chemicals (Table A-2-21). Benthic invertebrates also serve as an important exposure route from contaminated sediment to higher-trophic-level organisms (Figure A-2-4; Sections A.2.2.3 and A.2.2.4). A COPC screen for crabs is presented in Section A.7.1.1.2 based on exposure and effects data presented in Sections A.3.1.2.1 and A.3.2.4.

Table A-2-21. COPCs retained for benthic invertebrates [a,b,c,d]

Retained Due to Measured Concentration Greater than SQS or SL			Retained due to Detection Limit Greater than SQS or SL
1,2,4-Trichlorobenzene	Butyl benzyl phthalate	Naphthalene	1,4-Dichlorobenzene[f]
1,2-Dichlorobenzene	Cadmium	Nickel[e]	2-Methylphenol[f]
1,3-Dichlorobenzene[e]	Chlordane, alpha[e]	N-Nitrosodiphenylamine[e]	Acenaphthylene[f]
2,4-Dimethylphenol	Chromium	PCBs (total-calculated)	Aldrin[e,f]
2-Methylnaphthalene	Chrysene	Pentachlorophenol	Diethylphthalate[f]
4-Methylphenol	Copper	Phenanthrene	Dimethyl phthalate[f]
Acenaphthene	Dibenzo(a,h)anthracene	Phenol	Di-n-octyl phthalate[f]
Anthracene	Dibenzofuran	Pyrene	Ethylbenzene[e,f]
Arsenic	Dieldrin[e]	Silver	Gamma-BHC[e,f]
Benzo(a)anthracene	Di-n-butylphthalate	Tributyltin[b]	Heptachlor[e,f]
Benzo(a)pyrene	Fluoranthene	Total DDTs (calculated)[e]	Hexachlorobutadiene[e,f]
Benzo(g,h,i)perylene	Fluorene	Total HPAH (calculated)	Hexachloroethane[e,f]
Benzofluoranthene (total)	Hexachlorobenzene	Total LPAH (calculated)	Tetrachloroethene[e,f]
Benzoic acid	Indeno(1,2,3-cd)pyrene	Zinc	Trichloroethene[e,f]
Benzyl alcohol	Lead		
Bis(2-ethylhexyl)phthalate	Mercury		

[a] COPCs retained based on a comparison between maximum sediment concentrations and SMS sediment quality standards (SQS) and DMMP screening levels (SL)

[b] TBT does not have a bulk sediment-based SQS or SL, and was screened in based on the Weston (1999) analysis

[c] Of the DMMP chemicals, only antimony and xylene were screened out.

[d] No COPCs were screened out for crab in the problem formulation. All available tissue and effects data for crab were evaluated in the exposure and effects assessments as well as the risk characterization.

[e] Analyte screened using DMMP SL because no SQS was available

[f] Analyte had detection limit greater than SQS or SL (when SQS is not available)

Fish are primarily exposed to COPCs in sediment through direct dermal contact with sediment (English sole), incidental ingestion of sediment during feeding (English sole), and feeding on contaminated prey (Figure A-2-4). COPCs for juvenile salmonids,

English sole, and bull trout are listed in Table A-2-22, based on the screens conducted in Section A.2.4.6. Fish serve as a major route of food-chain transfer because they are prey for other fish, birds, and mammals (Figure A-2-4).

Birds and mammals are primarily exposed through food-chain transfer, although most species, especially spotted sandpiper, are also exposed directly to sediment during foraging (Figure A-2-3). COPCs for spotted sandpiper, great blue heron, bald eagle, river otter, and harbor seal are listed in Table A-2-22 based on the screens described in Section A.2.4.7.

Emergent aquatic plants are exposed to sediment-associated chemicals directly through their contact with sediment. Plants serve important functions in the estuary, including primary production and creation of important habitat for a number of species (Section A.2.2.5). COPCs for emergent aquatic plants are listed in Table A-2-22 based on the screen presented in Section A.2.4.8.

Table A-2-22. ROC/COPC pairs to be evaluated in the exposure and effects assessments for fish, wildlife, and plants

	PCBs	PAHs	TBT	BEHP	DDTs	As	Cu	Pb	Zn	Hg
Juvenile chinook salmon	×	×	×	[a]	×	×	×	[a]	[a]	×
English sole	×	×	×	[a]	×	×	×	[a]	[a]	×
Bull trout	×	[d]	×	[a]	×	×	×	[a]	[a]	×
Sandpiper	×	[a]	[a]	×	[b, e]	[a]	×	×	×	[a]
Heron	×	[a]	[a]	[a]	[a]	[a]	[a]	×	[a]	×
Eagle	×	[a]	[a]	[a]	[e]	[a]	[a]	×	[a]	×
Otter	×	[a]	[a]	[a]	[a]	×	[a]	×	[a]	[a]
Seal	×	[e]	[a]	[a]	[a]	[a]	[a]	[a]	[a]	[a]
Emergent aquatic plants	×	[a]	[c]	[a]	[c]	[a]	[a]	×	×	×

BEHP – bis(2-ethylhexyl)phthalate

DDTs – sum of DDT, DDE, and DDD

[a] ROC/COPC pair screened out because maximum potential exposure concentrations were less than NOEC (concentration) or NOAEL (dose) toxicity data.

[b] ROC/COPC pair not screened due to lack of exposure data.

[c] ROC/COPC pair not screened due to lack of effects data.

[d] ROC/COPC pair not further evaluated due to an incomplete exposure pathway.

[e] ROC/COPC pair not further evaluated in the Phase 1 ERA (HQ<1), but the feasibility and utility of collecting additional exposure data are discussed in the data gaps memorandum.

A.2.5.4 Assessment endpoints and measures of effect and exposure

Assessment endpoints are defined as the explicit expressions of the ecological resources that are to be protected (EPA 1992). These resources include those vital to ecosystem function, those providing critical resources such as habitat and fisheries, and those perceived as valuable by humans such as threatened or endangered (T&E) species and other issues addressed by legislation. They must define both the valued

entity and the characteristic of the entity to be protected. They provide direction for the risk assessment and are the basis for the analyses. Unless an ecological receptor is listed as a T&E species, assessment endpoints are selected that are relevant to population-level rather than individual effects. For threatened and endangered species, risks to individuals are important to evaluate (EPA 1998), although specific guidance regarding this approach is not available. Other EPA Region 10 Superfund sites, such as Coeur d'Alene and Blackbird Mine, have placed greater emphasis on NOECs than on LOECs for the protection of T&E species.

The selection of assessment endpoints was based on available information regarding the ecological relevance of the endpoint and on societal values. In addition, assessment endpoints were evaluated to ensure that their protection would likely result in protection of other valued entities within the system. Finally, endpoints selected must be amenable to assessment either through existing data or data that may be collected in the next phase of the RI. Only those ROC/COPC combinations with complete and significant exposure pathways were selected.

Assessment endpoints for each ROC are listed in Table A-2-23 along with measures of exposure and effect used in the exposure and effects assessments (Sections A.3 through A.6). Survival, growth, and reproduction are the key endpoints under review for most species in this assessment. Potential effects on survival attributable to immunosuppression are also included for juvenile salmonids and harbor seals based on existing data and the need for a careful assessment of existing data. Biomarker and histological endpoints are not included as assessment endpoints. Typically, ERAs focus on ecological effects at the individual level or higher (i.e., population level). In this way, the emphasis is placed on endpoints that integrate an overall response by an organism, rather than indicators of a biochemical response that may or may not result in an ecologically relevant effect. Research is ongoing in the area of biomarkers to better understand their significance for potential use in ERA. For biomarkers to be useful in determining sediment-associated risk, they must have clear dose-response data relating exposure to ecologically significant effects. Other responses, such as biliary FACs and DNA adducts, are categorized as a measure of exposure rather than as an assessment endpoint. Exposure data, including those for relevant biomarkers, will be presented in the exposure and effects assessment. Biomarker data will be discussed in the uncertainty assessment (Section A.7.2.2). Lesion data for English sole, and their potential to have population-level effects due to mortality, are examined in the effects assessment, Section A.4.3.

Table A-2-23. Assessment endpoints for ROCs and measures of effect and exposure

ROC	Assessment Endpoint	Measures of Effect and Exposure
Benthic		
The benthic community	Survival, growth, reproduction	Comparison of surface sediment concentrations to SMS and DMMP guidelines; evaluation of site-specific toxicity tests and community analyses; comparison of TBT tissue concentrations to effects data from the literature
Crab	Survival, growth, reproduction	Comparison of tissue data to residue-based effects data from the literature
Fish		
Juvenile chinook salmon	Survival (and immunocompetence) and growth	Comparison of juvenile salmon exposure data based on modeled or measured concentrations of chemicals in sediments, fish tissues, and prey tissues to relevant toxicological data
English sole	Survival, growth, reproduction	Comparison of English sole exposure data based on modeled or measured concentrations of chemicals in sediments, fish tissues, and prey tissues to relevant toxicological data
Bull trout	Survival, growth, reproduction [a]	Comparison of bull trout exposure data based on modeled or measured concentrations of chemicals in sediments, fish tissues, or prey tissues to relevant toxicological data
Wildlife		
Great blue heron	Survival, growth, reproduction	Modeled site-specific exposure and measured concentrations of chemicals in heron eggs will be compared to TRVs
Bald eagle	Survival, growth, reproduction	Modeled site-specific exposure will be compared to TRVs
Spotted sandpiper	Survival, growth, reproduction	Modeled site-specific exposure will be compared to TRVs
River otter	Survival, growth, reproduction	Modeled site-specific exposure will be compared to TRVs
Harbor seal	Survival (and immunocompetence), growth, reproduction	Modeled site-specific exposure will be compared to TRVs
Plants		
Emergent aquatic plants	Survival, growth, development	Comparison of emergent aquatic plant exposure based on concentrations of chemicals in marsh sediments to background concentrations and relevant toxicological data.

[a] For non-biomagnifying COPCs, the only reproductive endpoints that will be assessed are those relevant to bull trout, such as maternal transfer. Because spawning by bull trout does not occur in the LDW, exposures that involve eggs and larvae are not relevant. However, for biomagnifying COPCs, all reproductive endpoints will be assessed.

A.3 Exposure and Effects Assessment: Benthic Invertebrates

Benthic invertebrates are a diverse community of year-round resident organisms that live in and on the sediment and other substrates. In the LDW, this community includes crustaceans, oligochaetes, polychaetes, echinoderms, nematodes, and bivalves (Table A-2-2). The entire benthic invertebrate community as a whole is being evaluated as an ROC in this Phase 1 ERA (see Section A.2.3.1). Also, because some

epibenthic invertebrates are more mobile than most benthic infauna and are also higher on the food web, crab was also selected as an ROC. Crabs represent epibenthic invertebrates not specifically addressed by the available sediment quality standards and guidelines,[50] which are intended to protect relatively sessile benthic fauna. Because of its mobility, a crab's exposure to sediment-associated chemicals is integrated over a wider area. Consequently, a tissue residue approach was used to evaluate exposure for crabs, rather than sediment chemistry data.

COPCs for the benthic invertebrate community were identified in the problem formulation (Section A.2.4.5) by comparing maximum sediment concentrations from the LDW to the SQS of the SMS[51] or the DMMP[52] screening level (SL),[53] for chemicals without a SQS. Fifty-nine chemicals were retained as COPCs based on the SQS/SL screen. TBT was also added as a COPC for benthic infauna in the problem formulation (Section A.2.4.5).

The remainder of this section is divided into an exposure assessment (Section A.3.1) and an effects assessment (Section A.3.2) to assess the COPCs previously identified in Section A.2.4.5. The potential exposure of benthic infaunal invertebrates to these COPCs was evaluated through a quantitative and spatial analysis of surface sediment chemistry data (Section A.3.1.1), except for TBT, which was analyzed using a tissue residue approach (Section A.3.1.2). Exposure of other benthic invertebrates, as represented by crabs, to COPCs was evaluated using a tissue residue approach in Section A.3.1.2.

In Section A.3.2, potential effects data associated with chemical concentrations in sediment and tissue are presented. The effects data for infaunal benthic invertebrates are primarily the SMS, which are based on Puget Sound sediment AETs, and are presented in Section A.3.2.1. The effects assessment also presents the limited available site-specific sediment toxicity (Section A.3.2.2) and benthic community data (Section A.3.2.3). Effects data for crabs and TBT effects data for benthic invertebrates are tissue-based; these data are presented in Sections A.3.2.4 and A.3.2.5, respectively.

---

[50] Hereafter in this document, the general term "sediment quality standards" refers to both the SQS and CSL, and the term "guidelines" refers to DMMP SL and ML, unless otherwise noted.

[51] SQS (WAC 173-204-320) are part of the Washington State Sediment Management Standards (SMS)

[52] *Dredged Material Evaluation and Disposal Procedures - A Users Manual for the Puget Sound Dredged Disposal Analysis (PSDDA) Program.* February 2000)

[53] The SQS and SLs are both based on Puget Sound Apparent Effects Threshold (AET) values (PTI 1988), but they are used for different purposes. The SQS have been promulgated as state regulations and are used much like water quality criteria to assess marine sediment quality. The SLs are used to determine the suitability of dredged material for open-water disposal. Conceptually, the SMS standards and DMMP guidelines each provide two regulatory levels for the evaluation of sediment contaminant concentrations. The SQS under the SMS and the SLs under the DMMP program represent concentrations below which adverse biological effects are considered to be unlikely. The Cleanup Screening Level (CSL) under the SMS and the Maximum Level (ML) under the DMMP represent concentrations above which adverse biological effects are predicted to be significant and further actions are required.

The exposure and effects data presented in this section are combined in the risk characterization (Section A.7.1).

A.3.1 EXPOSURE ASSESSMENT

The exposure assessment for infaunal benthic invertebrates is based primarily on the nature and extent of chemical concentrations in sediment inhabited by these animals. As previously discussed in the problem formulation, over 1,000 surface sediment samples have been collected in the LDW and analyzed for COPCs within the last 10 years[54], resulting in a large body of information that can be used to characterize the chemical exposure regime for infaunal invertebrates (Section A.3.1.1). As previously discussed in the problem formulation, the exposure assessment for crabs (Section A.3.1.2.1) is based on tissue chemistry data because existing sediment quality standards and guidelines are not applicable to these mobile higher-trophic-level animals. TBT tissue concentrations in amphipods are also presented as indications of exposure (Section A.3.1.2.2), because bulk sediment-based quality standards and guidelines have not been promulgated to assess the potential for adverse effects to benthic invertebrates from this chemical.

A.3.1.1 Sediment chemistry

In this section, sediment data for COPCs are presented to characterize the exposure regime for benthic invertebrates, and to illustrate the spatial distribution, magnitude, and frequency of sediment quality standard or guideline exceedances. The objective of the sediment chemistry analysis is to further evaluate chemicals with concentrations that are elevated relative to sediment quality standards or guidelines.

The LDW RI SOW requires that sediment chemistry data be assessed relative to sediment quality standards. Comparing site-specific sediment chemistry data with SMS standards or DMMP guidelines expresses the potential extent of benthic invertebrate exposure to sediment-associated chemicals. It is not the intent of this section to imply risk based on concentrations identified above or below the SQS or SL; that characterization is provided in Section A.7.1.

Two types of analyses are presented in this section. First, historical surface sediment chemistry data are compared to applicable sediment quality standards and guidelines in greater detail than was presented previously in the problem formulation, which primarily focused on maximum chemical concentrations. Second, to better understand the spatial distribution of sediment-associated chemicals, results of a series of geographic information system (GIS) analyses are presented. These analyses were performed to:

[54] Data from the Harbor Island Remedial Investigation were collected more than 10 years ago. For the sake of continuity throughout the project, they are being used in the risk assessment because the data set was identified as a suitable data source at the beginning of the project.

- determine the spatial distribution of COPCs identified in the problem formulation
- evaluate whether COPC exceedances are co-located (i.e., single locations with exceedances of more than one chemical)

A.3.1.1.1 Quantitative analysis of chemicals of potential concern

As previously discussed, chemicals were identified as COPCs for benthic invertebrates in the problem formulation (Section A.2.4.5) if maximum sediment chemical concentrations[55] were greater than the applicable SQS (or SL, if an SQS was unavailable). Fifty-nine chemicals with sediment quality standards or guidelines were retained as COPCs based on the initial SQS/SL screen. Antimony and total xylenes were the only chemicals whose maximum detected concentration or detection limit did not exceed the SQS or SL. There were 274 additional chemicals without either an SQS or SL that were not retained as COPCs. The effect of these chemicals on the assessment of risk will be discussed in the uncertainty assessment (Section A.7.1.2).

Due to the large number of benthic COPCs identified in the problem formulation, a three-step decision process (Figure A-3-1) was developed to help focus the exposure discussion and the subsequent sections of the Phase 1 risk assessment (i.e., risk characterization and uncertainty analysis). Note, however, that all chemicals with sediment quality standard or guideline exceedances are considered in the overall risk characterization for benthic invertebrates (see Section A.7.1). A 5% frequency of detection threshold was selected for several steps in the decision process so that infrequently detected chemicals, or chemicals whose concentrations rarely exceeded standards or guidelines, could be characterized in less detail in subsequent sections of the risk assessment, thereby focusing attention on chemicals most likely to pose risk to benthic invertebrates. This grouping process is consistent with EPA risk assessment guidelines for focusing risk assessments when a large number of chemicals need characterization (EPA 1989).

Step 1 identified chemicals detected at more than 5% of the locations. In Step 2a, the chemicals selected in Step 1 were re-screened to identify those for which more than 5% of the detected concentrations exceeded SQS or SL. These two steps thus identify chemicals for which there are multiple detections and exceedances of standards or guidelines.

[55] If chemicals were analyzed, but not detected, their maximum detection limits were compared to applicable guidelines.

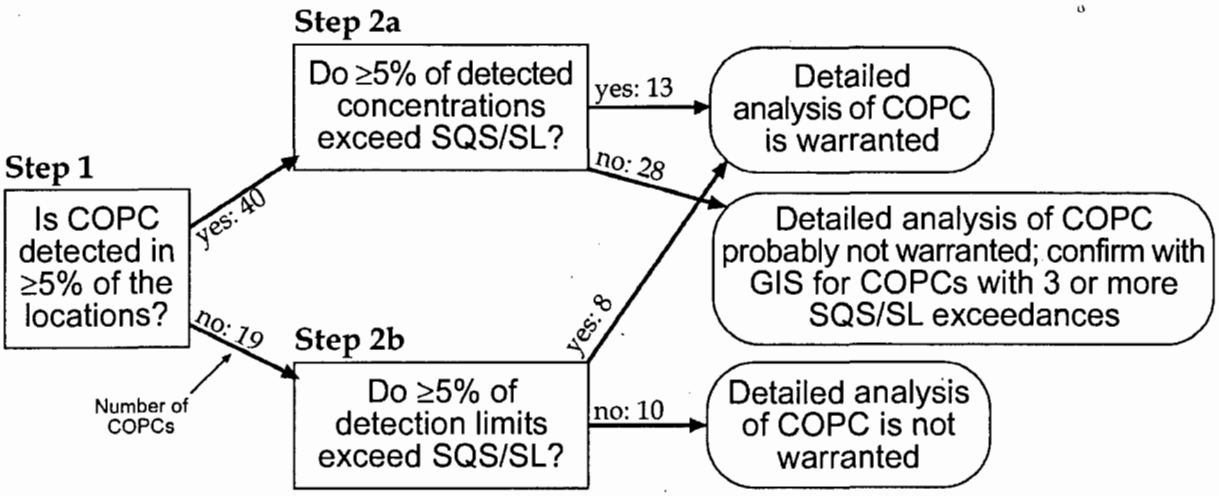

Figure A-3-1. Decision process for focusing benthic invertebrate COPC list for additional analysis in the exposure assessment

Chemicals identified in Step 1 as being detected at fewer than 5% of the locations were re-examined in Step 2b to identify those with detection limits above standards or guidelines. Chemicals with detection limits greater than standards or guidelines were re-examined because it is possible they could have exceeded standards or guidelines more frequently had detection limits been lower. Step 2b identified chemicals for which more than 5% of locations had detection limits above the SQS or SL. The chemicals identified in Step 2b were then grouped with chemicals identified from Step 2a as warranting further detailed analysis in the exposure assessment.

Chemicals screened out in Step 2a because fewer than 5% of the detected concentrations exceeded standards or guidelines were re-examined in a GIS confirmatory step. The GIS evaluation was intended to identify chemicals that rarely exceeded standards or guidelines, but may result in a station group[56] if the few exceedances were located in a small area. The results of the analysis are presented later in this section.

Application of the decision process shown in Figure A-3-1 yielded five COPC groups, as defined below:

- **COPC Group 1**—Detection frequency ≥ 5% (yes for Step 1) and SQS/SL exceedance frequency ≥ 5% (yes for Step 2a) - 13 chemicals
- **COPC Group 2**—Detection frequency ≥ 5% (yes for Step 1), SQS/SL exceedance frequency < 5% (no for Step 2a), detailed GIS analysis warranted because there were 3 or more exceedances by detected concentrations - 20 chemicals[57]

[56] Station group defined as three or more stations in close proximity, all of which had SQS/SL exceedances

[57] Group 2 represents 20 of the 28 chemicals resulting from a "no" answer in Figure A-3-1 Step 2a

- **COPC Group 3**—Detection frequency ≥ 5% (yes for Step 1), SQS/SL exceedance frequency < 5% (no for Step 2a), detailed GIS analysis not warranted because there were less than 3 exceedances - 8 chemicals [58]
- **COPC Group 4**—Detection frequency < 5% (no for Step 1), but SQS/SL exceedance frequency for detection limits > 5% (yes for Step 2b) - 8 chemicals
- **COPC Group 5**—Detection frequency < 5% (no for Step 1) and SQS/SL exceedance frequency for detection limits < 5% (no for Step 2b) - 10 chemicals

The data used to identify the chemicals for each of the decision process steps outlined above in Figure A-3-1 are summarized in Table A-3-1. The summary statistics presented in this table include the number of locations where the COPC was analyzed and detected and a comparison of the detected concentration and detection limits with standards or guidelines. In Table A-3-1, COPCs were organized according to the five COPC groups defined above. Each location-specific concentration was compared to the applicable standard or guideline, creating the exceedance factors (EFs) shown in the table. The range of EFs is shown for each chemical to provide additional information about the distribution of chemical concentrations, but not necessarily the relative degree of risk, in comparison to standards or guidelines. EFs have no regulatory relevance. The numbers of locations and the EFs were calculated separately for detected concentrations and detection limits (nondetects) to emphasize the difference between an exceedance based on a detected concentration and one based on an analytical detection limit.

The results of the decision process described in Figure A-3-1, using the data in Table A-3-1, are discussed below.

Detection frequencies were ≥ 5% for 40 of the 59 COPCs (Step 1). Of the 40 COPCs identified from Step 1, 13 were also identified for further analysis from Step 2a (i.e., ≥ 5% detected concentrations exceeded SQS or SL) and were assigned to COPC Group 1.

GIS analysis was conducted on COPCs identified in Step 2a as having very low (i.e., < 5%) SQS/SL exceedance frequencies with at least three exceedances to confirm that infrequent exceedances were not located in an isolated area within the LDW that could be defined as a station group. At least three exceedances were necessary to define a station group; therefore, GIS confirmatory analysis was applied only to COPCs whose detected concentrations exceeded SQS/SLs in three or more samples.
Twenty chemicals had at least three SQS/SL exceedances and were subject to confirmatory GIS analysis and assigned to COPC Group 2. The remaining 8 chemicals had less than 3 SQS/SL exceedances based on detected concentrations and were assigned to COPC Group 3.

[58] Group 3 represents 8 of the 28 chemicals resulting from a "no" answer in Figure A-3-1 Step 2a

Table A-3-1. Summary of chemical-specific exceedances of SQS or SL

Chemical	Detected Concentrations			Detection Limits (Non-detects)		
	# of Locations	# Locations Exceeding SQS or SL[a]	SQS or SL EF Range[b]	# of Locations	# Locations Exceeding SQS or SL[a]	SQS or SL EF Range[b]
COPC Group 1 – Detection frequency ≥ 5% and SQS/SL exceedance frequency ≥ 5%						
1,2-Dichlorobenzene	35	2 (5.7%)	0.020 - 4.8	522	85 (16%)	0.011 - 57
4-Methylphenol	36	6 (17%)	0.024 - 9.3	245	6 (2.4%)	0.010 - 3.1
Acenaphthene	229	23 (10%)	0.0044 - 11	328	11 (3.4%)	0.0037 - 5.6
Benzoic acid	30	3 (10%)	0.10 - 9.1	519	69 (13%)	0.020 - 3.1
Bis(2-ethylhexyl)phthalate	466	101 (22%)	0.0043 - 11	95	1 (1.1%)	0.015 - 2.9
Butyl benzyl phthalate	336	71 (21%)	0.014 - 110	225	54 (24%)	0.012 – 32
DDTs (total-calculated)[c, d]	42	21 (50%)	0.14 - 420	60	10 (17%)	0.13 - 7.4
Dibenzofuran	188	12 (6.4%)	0.0073 - 6.5	368	14 (3.8%)	0.0040 – 6.0
Fluorene	299	16 (5.4%)	0.0039 - 7.4	258	3 (1.2%)	0.0026 - 3.7
Hexachlorobenzene	41	5 (12%)	0.047 - 120	516	357 (69%)	0.025 - 240
Mercury*	501	27 (5.4%)	0.049 - 11	71	0	0.049 - 0.54
PCBs (total-calculated)[e]	905	345 (38%)	0.010 - 880	52	1 (1.9%)	0.0032 - 1.4
Phenol	197	14 (7.1%)	0.048 - 8.6	360	3 (0.83%)	0.029 - 4.8
COPC Group 2 – Detection frequency ≥ 5%, SQS/SL exceedance frequency < 5%, additional GIS analysis warranted because 3 or more exceedances by detected concentrations						
1,4-Dichlorobenzene	69	3 (4.3%)	0.0077 - 21	488	78 (16%)	0.0074 - 29
Arsenic*	525	4 (0.76%)	0.032 - 1.7	50	0	0.054 - 0.54
Benz(a)anthracene	511	9 (1.8%)	0.0041 - 7.3	46	0	0.0061 - 0.33
Benzo(a)pyrene	511	8 (1.6%)	0.0040 - 8.2	46	0	0.011 - 0.37
Benzo(g,h,i)perylene	489	14 (2.9%)	0.0071 - 17	68	8 (12%)	0.014 - 3
Benzofluoranthene (total)	511	7 (1.4%)	0.0050 - 5.4	39	0	0.0046 - 0.14
Cadmium*	430	11 (2.6%)	0.014 - 24	137	0	0.0078 - 0.31
Chromium*	571	7 (1.2%)	0.019 - 4.2	na	na	na
Chrysene	529	19 (3.6%)	0.0086 - 8.1	28	0	0.0067 - 0.19
Copper*	575	6 (1.0%)	0.013 - 31	na	na	na
Dibenz(a,h)anthracene	330	15 (4.5%)	0.0067 - 23	227	18 (7.9%)	0.016 - 8.7
Fluoranthene	540	26 (4.8%)	0.0058 - 15	17	0	0.0042 - 0.24
Indeno(1,2,3-cd)pyrene	492	20 (4.1%)	0.0082 - 17	65	6 (9.2%)	0.021 - 3.2
Lead*	575	14 (2.4%)	0.0044 - 51	na	na	na
Nickel*[c]	563	7 (1.2%)	0.036 - 6.5	2	0	0.21 - 0.23
Phenanthrene	520	22 (4.2%)	0.0036 - 17	37	0	0.0067 - 0.47
Silver*	408	8 (2.0%)	0.0066 - 44	159	0	0.033 - 0.54
Total HPAH (calculated)	544	16 (2.9%)	0.00087 - 9.7	13	0	0.0011 - 0.02
Total LPAH (calculated)	522	8 (1.5%)	0.0012 - 6.3	35	0	0.0028 - 0.069
Zinc*	573	27 (4.7%)	0.039 - 24	2	0	0.31 - 0.83
COPC Group 3 – Detection frequency ≥ 5%, SQS/SL exceedance frequency < 5%, additional GIS analysis not warranted because < 3 exceedances						
2-Methylnaphthalene	87	1 (1.1%)	0.0024 - 1.6	470	7 (1.5%)	0.0016 - 3
Acenaphthylene[f]	57	0	0.0018 - 0.077	500	6 (1.2%)	0.00091 - 3.6
Anthracene	401	1 (0.25%)	0.00045 - 1.6	156	1 (0.64%)	0.0020 - 2.1
Dimethyl phthalate	109	0	0.0071 - 0.22	452	8 (1.8%)	0.0011 - 28

Chemical	Detected Concentrations			Detection Limits (Non-detects)		
	# of Locations	# Locations Exceeding SQS or SL[a]	SQS or SL EF Range[b]	# of Locations	# Locations Exceeding SQS or SL[a]	SQS or SL EF Range[b]
Di-n-butyl phthalate	183	0	0.0015 - 0.63	378	3 (0.79%)	0.00027 - 1.4
Di-*n*-octyl phthalate[f]	43	0	0.0019 - 0.61	518	8 (1.5%)	0.0010 - 4.8
Naphthalene	91	1 (1.1%)	0.0019 - 1	466	0	0.00060 - 0.95
Pyrene	531	1 (0.19%)	0.00086 - 1.8	26	0	0.00067 - 0.22
COPC Group 4 – Detection frequency < 5%, but SQS/SL exceedance frequency for detection limits ≥ 5%						
1,2,4-Trichlorobenzene	7	1 (14%)	0.071 - 2.7	550	316 (57%)	0.031 - 110
2,4-Dimethylphenol	1	1 (100%)	10 - 10	552	165 (30%)	0.20 - 72
2-Methylphenol[d]	2	0	0.32 - 0.92	555	79 (14%)	0.094 - 33
Alpha-chlordane[c]	1	1 (100%)	2.6 - 2.6	54	5 (9.3%)	0.081 - 3.7
Benzyl alcohol	7	3 (43%)	0.40 - 30	542	80 (15%)	0.10 - 12
Hexachlorobutadiene[c,f]	nd	nd	nd	557	101 (18%)	0.015 - 180
N-Nitrosodiphenylamine[f]	8	0	0.22 - 0.7	549	40 (7.3%)	0.0054 - 71
Pentachlorophenol	5	1 (20%)	0.28 - 1.5	501	83 (17%)	0.019 - 14
COPC Group 5 – Detection frequency < 5% and SQS/SL exceedance frequency for detection limits < 5%						
1,3-Dichlorobenzene [c]	9	0	0.0049 - 0.58	541	10 (1.8%)	0.0021 - 12
Aldrin[c,e]	nd	nd	nd	100	2 (2.0%)	0.046 - 5.6
Dieldrin [c]	5	3 (60%)	0.31 - 28	95	5 (5.3%)	0.063 - 5.6
Diethyl phthalate[f]	8	0	0.021 - 0.33	553	8 (1.4%)	0.00098 - 42
Ethylbenzene [c,d]	1	0	0.049 – 0.049	48	3 (6.3%)	0.14 - 53
Gamma-BHC[c,f]	3	0	0.49 - 0.86	97	2 (2.1%)	0.046 - 5.6
Heptachlor[c,f]	4	0	0.18 - 0.52	96	2 (2.1%)	0.063 - 5.6
Hexachloroethane[c,f]	nd	nd	nd	546	4 (0.73%)	0.0011 - 1.5
Tetrachloroethene[c,f]	2	0	0.0037 - 0.0091	47	1 (2.1%)	0.025 - 9.4
Trichloroethene[c,f]	nd	nd	nd	49	1 (2.0%)	0.0088 - 3.3

[a] Numbers in parentheses are precent of locations exceeding SQS or SL

[b] SQS EF = measured concentration ÷ SQS; or measured concentration ÷ SL (when SQS not available). Note that the EF has no regulatory relevance, and is presented here to indicate the relative magnitude of measured concentrations or detection limits.

[c] Analyte screened using DMMP SL instead of SQS. TBT was assessed using a tissue approach, so it is not presented in this table.

[d] The sum of detected concentrations for p,p'-DDD, p,p'-DDE, and p,p'-DDT. In the event that all three compounds were undetected in a given sample, the total is equal to the highest detection limit among the three compounds.

[e] The sum of detected concentrations for 7 Aroclors (all sampling events except NOAA site characterization) or calculated as the sum of total PCBs and total polychlorinated terphenyls (PCTs) minus total PCTs (NOAA site characterization data). In the event that all Aroclors were undetected in a given sample, the total is equal to the highest detection limit among the 7 Aroclors.

[f] Identified as a COPC based on detection limit exceedance of SQS or SL

Units: all units are µg/kg dry weight (dw), except for metals (marked with an asterisk), which are mg/kg dw

* Metals

nd – not detected

na – not applicable

Section A.3.1.1.2 presents the GIS spatial analysis for the 20 COPC Group 2 chemicals. All 20 COPC Group 2 chemicals were detected at hundreds of surface sediment locations throughout the LDW (Table A-3-1); however, none of the few exceedances based on detected values constituted a station group. Given the relatively high

sampling density and detection frequency, and the relatively low exceedance frequency for these chemicals, it is unlikely that any large hot spots remain undiscovered. Consequently, Group 2 chemicals were not considered a high priority for additional analysis in Phase 1. These chemicals may be re-evaluated in Phase 2. Group 2 chemicals are included in the depiction of SQS exceedences (see Appendix A map folio).

Detection frequency was less than 5% for 19 of the 59 COPCs (Step 1). Of these 19 chemicals, 8 had ≥ 5% of their detection limits exceeding SQS/SL (step 2b) and were assigned to COPC Group 4 for detailed analysis. Note, however, that characterizing exposure to rarely detected chemicals using their detection limit is uncertain because it is not known if actual concentrations exceed standards or guidelines. Ten chemicals were assigned to COPC Group 5 because they were detected in less than 5% of the locations and detection limits exceeded SQS/SL in less than 5% of the locations.

The decision process represented in Figure A-3-1 identified 21 COPCs (COPC Groups 1 and 4) for detailed analysis. These COPCs included PCBs, mercury, two chlorinated pesticides, two PAHs, two phthalates, four chlorinated benzenes, phenol and four phenol derivatives, and five miscellaneous organic compounds (Table A-3-1). COPCs in COPC Groups 2, 3, and 5 were included in the comprehensive GIS analysis described below for all COPCs (Section A.3.1.1.2), but are not further discussed individually in this document. The remainder of the quantitative analysis presented in this section focuses on COPCs in COPC Groups 1 and 4.

Although the SQS or SL was used to assign membership to the five COPC groups described above, comparison to the CSL or ML provides additional perspective on the magnitude of COPC exposure. Table A-3-2 presents summary statistics for the comparison between surface sediment concentrations and CSL/ML for the 21 COPCs in COPC Groups 1 and 4. Although most of the 21 COPCs exceeded the CSL or ML at one or more locations, detected concentrations for 18 of the 21 COPCs in COPC Groups 1 and 4 exceeded the CSL or ML at fewer than 10 locations (Table A-3-2). PCBs, bis(2-ethylhexyl)phthalate (BEHP), and mercury exceeded the CSL at 134, 59, and 13 locations, respectively. In Group 1, concentrations of 5 of the 13 chemicals (PCBs, DDTs, BEHP, 1,2-dichlorobenzene, and 4-methylphenol) exceeded the CSL or ML at more than 10% of the locations at which they were detected. 4-Methylphenol was detected infrequently, however.

Table A-3-2. Summary of chemical-specific exceedances of CSL or ML for COPCs that warranted detailed analysis (COPC Groups 1 and 4)

	Detected Concentrations			Detection Limits (Non-detects)		
Chemical	# Samples	# Locations Exceeding CSL or ML [a]	CSL or ML EF Range [b]	# Samples	# Locations Exceeding CSL or ML [a]	CSL or ML EF Range [b]
COPC Group 1 – Detection frequency ≥ 5% and SQS/SL exceedance frequency ≥ 5%						
1,2-Dichlorobenzene	35	2 (5.7%)	0.020 - 4.8	522	85 (16%)	0.011 - 40
4-Methylphenol	36	6 (17%)	0.024 - 9.3	245	6 (2.4%)	0.010 - 3.1
Acenaphthene	229	3 (1.3%)	0.0012 - 3	328	3 (0.91%)	0.0011 - 1.7
Benzoic acid	30	3 (10%)	0.10 - 9.1	519	69 (13%)	0.020 - 3.1
BEHP	466	59 (13%)	0.0026 - 6.5	95	1 (1.1%)	0.011 - 1.7
Butyl benzyl phthalate	336	6 (1.8%)	0.0011 - 8.3	225	7 (3.1%)	0.00093 - 4.3
DDTs (total calculated) [c]	42	6 (14%)	0.014 - 42	60	0	0.013 - 0.74
Dibenzofuran	188	2 (1.1%)	0.0019 - 1.7	368	3 (0.82%)	0.0010 - 1.7
Fluorene	299	4 (1.3%)	0.0011 - 2.1	258	0	0.00076 - 0.36
Hexachlorobenzene	41	1 (2.4%)	0.0078 - 19	516	88 (17%)	0.0041 - 39
Mercury	501	13 (2.6%)	0.034 - 7.8	71	0	0.034 - 0.37
PCBs (total-calculated)	905	134 (15%)	0.0016 - 160	52	0	0.00059 - 0.26
Phenol	197	4 (2.0%)	0.017 – 3.0	360	1 (0.28%)	0.010 - 1.7
COPC Group 4 – Detection frequency < 5%, but SQS/SL exceedance frequency for detection limits ≥ 5%						
1,2,4-Trichlorobenzene	7	1 (14%)	0.032 - 1.2	550	105 (19%)	0.014 - 50
2,4-Dimethylphenol	1	1 (100%)	1.4 - 1.4	552	165 (30%)	0.020 - 72
2-Methylphenol	2	0	0.32 - 0.92	555	79 (14%)	0.094 - 33
Alpha-chlordane [d]	1	0	na	54	5 (9.3%)	0.081 - 3.7
Benzyl alcohol	7	3 (43%)	0.32 - 23	542	75 (14%)	0.081 - 9.5
Hexachlorobutadiene [c]	nd	nd	nd	557	77 (14%)	0.0096 - 17
N-Nitrosodiphenylamine	8	0	0.22 - 0.70	549	25 (4.6%)	0.0054 - 50
Pentachlorophenol	5	0	0.14 - 0.76	501	17 (3.4%)	0.0097 - 7.5

[a] Numbers in parentheses are precent of locations exceeding CSL or ML

[b] CSL EF = measured concentration ÷ CSL; or measured concentration ÷ ML (when CSL is not available). Note that the ratio has no regulatory relevance, and is presented here to provide an indication of the magnitude of measured concentrations or detection limits.

[c] Analyte screened using DMMP ML instead of CSL

[d] CSL or ML not available; data presented based on SL evaluation as presented in Table A-3-1

nd – not detected

na – not applicable

Tables A-3-3a (Group 1 COPCs) and A-3-3b (Group 4 COPCs) present the information previously provided in Table A-3-2 in rank order according to CSL (or ML) exceedance frequency. Detected concentrations are shown for Group 1 COPCs and detection limits are shown for Group 4 COPCs to reflect the rationale for their grouping as described in Figure A-3-1. Total PCBs and total DDTs had the highest maximum EFs for a single location (Table A-3-2).

For Group 1 COPCs (Table A-3-3a), detected concentrations exceeded the CSL/ML in 20% or fewer locations. While 4-methylphenol and DDTs had two of the highest

exceedance frequencies, they were based on a limited number of locations (i.e., 6 locations with detected concentrations) (Table A-3-2). PCB and BEHP had the other two highest exceedance frequencies, which were based on exceedances at 134 and 59 locations, respectively.

Table A-3-3a. Group 1 COPCs ranked by CSL/ML exceedance frequency

Chemical	CSL or ML Exceedance Frequency for the Detected Concentrations (%)
4-Methylphenol	17
PCBs (total-calc'd)	15
DDTs (total-calc'd)	14
BEHP	13
Benzoic acid	10
1,2-Dichlorobenzene	5.7
Mercury	2.6
Hexachlorobenzene	2.4
Phenol	2.0
Butyl benzyl phthalate	1.8
Fluorene	1.3
Acenaphthene	1.3
Dibenzofuran	1.1

The CSL/ML exceedance frequencies for Group 4 COPCs (Table A-3-3b) were based on 20 or more locations with detection limits above CSL/ML, except for pentachlorophenol and alpha-chlordane. 2,4-dimethylphenol, which had the highest frequency of detection limits exceeding CSL/ML (at more than 150 locations), also had the highest CSL EFs (72) for a detection limit (Table A-3-2). The uncertainty associated with the risk characterization using detection limits above the CSL/ML is discussed in Section A.7.1.2.

Table A-3-3b. Group 4 COPCs ranked by CSL/ML exceedance frequency

Chemical	CSL or ML Exceedance Frequency for the Detection Limits (%)
2,4-Dimethylphenol	30
1,2,4-Trichlorobenzene	19
2-Methylphenol	14
Hexachlorobutadiene	14
Benzyl alcohol	14
Alpha-chlordane [a]	9.3
N-Nitrosodiphenylamine	4.4
Pentachlorophenol	3.4

[a] CSL or ML not available; data presented based on SL

A.3.1.1.2 Spatial analysis

This section presents chemical-specific spatial analyses of SQS/SL and CSL/ML exceedances and is focused primarily on the chemicals listed in Table A-3-3a (COPC Group 1). Two of the COPCs listed in Table A-3-3a are of higher priority for spatial analysis because the number of CSL exceedances is much larger than other COPCs. Total PCBs (134 CSL exceedances for detected concentrations) and BEHP (59 CSL exceedances) were measured at elevated concentrations at multiple locations in the LDW. CSL exceedances for these two chemicals were only occasionally co-located (see Section A.7.1). CSL exceedances for the other 11 Group 1 COPCs were typically co-located with CSL exceedances of either BEHP or total PCBs, although there were some exceptions.

The spatial distribution of SQS/SL and CSL/ML exceedances is shown for four of the 13 Group 1 chemicals (total PCBs, BEHP, mercury, and total DDTs) in Maps A-3-1 to A-3-4 (Attachment A.1). These COPCs were mapped because they had the highest number of SQS exceedances or frequency of exceedance. There were fewer than 40 locations where standards or guidelines were exceeded for mercury or total DDTs, or any other COPC Group 1 chemical, that did not also exceed total PCBs or BEHP standards (Section A.7.1). Chemicals that were not individually shown in figures were included in maps that summarize SQS/SL (Map A-3-5) and CSL/ML (Map A-3-6) exceedances for all COPCs simultaneously.

Exceedances shown on the maps were represented as point locations (e.g., Map A-3-1a) and as Thiessen polygons (e.g., Map A-3-1b) for each chemical. Thiessen polygons are a method commonly used in spatial analysis to account for spatial variability in sampling intensity. The Thiessen polygon associates each point in a plane with the closest sampling location for which a measurement is available (Burmaster and Thompson 1997). In effect, this algorithm assumes that the concentration at any point where measurements have not been made is the same as the concentration in the sample closest to that point. This data display method can also be used to calculate area-weighted concentrations. On the point location maps, detection limit exceedances were shown separately from exceedances based on detected concentrations. To simplify the visual impact, the same distinction was not made on the Thiessen polygon maps because very few of the exceedances noted for these four chemicals were based on detection limits.

The spatial exceedance patterns for total PCBs, BEHP, mercury, and total DDTs are discussed below using river miles for reference, as shown on the maps. The remaining Group 1 COPCs (butyl benzyl phthalate, hexachlorobenzene, 1,4-dichlorobenzene, 4-methylphenol, benzoic acid, dibenzofuran, 1,2-dichlorobenzene, acenaphthene, fluorene, and phenol) showed no station groups based on detected concentrations and were not mapped. These chemicals are further evaluated in Section A.7.1 using all available surface sediment chemistry data.

Total PCBs

Total PCB concentrations in excess of the SQS or the CSL were found at various locations scattered throughout the LDW (Maps A-3-1a and A-3-1b). Areas where exceedances were most concentrated included the east side of the waterway from River Mile (RM) 0.3 to 0.6, from Slip 4 down to RM 4.0 (primarily on east side), and upstream of Turning Basin 3 at RM 4.7. Several CSL exceedances were found on the west side between RM 2.0 and 2.3 and between RM 3.5 and 3.7. Approximately 78% of the total LDW area was below the PCB SQS (based on Thiessen polygons), and 96% of the total area was below the PCB CSL.

Bis(2-ethylhexyl)phthalate

BEHP exceedances of the SQS or the CSL were found in the majority of samples collected on the east side between RM 0.3 and 0.6 (Maps A-3-2a and A-3-2b). Less frequent exceedances were found between Slips 4 and 6 (between RM 3.3 and 3.9). Isolated CSL exceedances were found at several other locations, but none of these areas would be categorized as station groups (i.e., three or more polygons in close proximity with CSL exceedances) for this chemical. BEHP concentrations in 93% of the total LDW area were below the SQS (based on Thiessen polygons), and concentrations in 97% of the total area were below the CSL.

Mercury

The distribution of mercury exceedances is shown in Maps A-3-3a and A-3-3b. Several CSL exceedances were found between RM 3.5 and 3.6. Other CSL exceedances were found between RM 0.4 and 0.6, at RM 1.4, at RM 2.2, and at Slip 4 (RM 2.8); however, these samples were interspersed among many other samples where no exceedances were found. Mercury concentrations in 97% of the total LDW area were below the SQS (based on Thiessen polygons).

Total DDT

Total DDT was analyzed at 102 locations in the LDW. Detected total DDT concentrations exceeded the ML at two locations and the SL at 12 additional locations between RM 0.4 and 0.6 (Maps A-3-4a and A-3-4b). Detected concentrations exceeded the ML at only four other locations and the SL at only three other locations scattered throughout the LDW. Total DDT concentrations (or detection limits) were below the SL in 76% of the total LDW area, and below the ML in 94% of the total area.

Other Analyses

In addition to the chemicals described above, 20 chemicals with detected concentrations that exceeded SQS/SL in three or more samples, but less than 5% of all surface sediment samples (Group 2 in Table A-3-1), were also evaluated using GIS to determine whether any COPCs formed a station group. No chemical-specific station groups were identified for these 20 chemicals.

In addition to the spatial distributions analyzed above, additional GIS analysis was performed to provide an overall indication of standard or guideline exceedances[59] based on the total number of chemicals that exceeded SQS/SL (Map A-3-5) and CSL/ML (Map A-3-6) at each location.

CSL/ML exceedances were found throughout the LDW, but were generally concentrated in several areas: RM 0.3 to 0.6 (east side), Slip 4, RM 3.3 to 3.6 (east side), and just upstream of Turning Basin 3 (RM 4.8 to 5.0). Isolated CSL/ML exceedances were found at several other areas, including south of Harbor Island (RM 0.1), Slip 3 and the west side opposite Slip 3 (RM 1.9 to 2.2), most of the east side between Slips 4 and 6, and downstream of RM 5.0 (Map A-3-6).

A.3.1.2 Tissue chemistry

This section assesses potential crab exposure to COPCs (Section A.3.1.2.1) and potential exposure of benthic invertebrates to TBT (Section A.3.1.2.2). Both assessments were conducted using tissue data collected within the LDW.

Crabs are epibenthic invertebrates associated with sediment, but are more mobile than many benthic invertebrates and are higher on the food web. These species are not specifically addressed by the available sediment quality standards or guidelines, which are intended to protect relatively sessile benthic invertebrates. Because of its mobility, a crab's exposure to sediment-associated chemicals is integrated over a wider area. Consequently, the tissue residue approach presented in this section was used to evaluate exposure for crabs, rather than a comparison of sediment chemistry data to available standards or guidelines.

TBT was also assessed from a tissue perspective because this approach is consistent with regional EPA precedence (EPA 1999) and other regional recommendations (Meador 2000). Thus, potential exposure of benthic invertebrate species to TBT was assessed using measured and estimated body burdens of TBT in benthic invertebrates.

A.3.1.2.1 Crab screen

This section presents available crab tissue data to assess potential exposure of sediment-associated COPCs to crabs that primarily inhabit the downstream, more saline portion of the LDW.

Crab tissue data from the LDW were available from two sampling events. In 1998, four samples of composited raw edible crabmeat were collected (two red rock crab samples, one Dungeness crab sample, and one combined red rock/Dungeness crab sample) and analyzed for mercury, PCBs, and TBT for human health risk evaluation. In 1997, King County collected two composite raw edible crabmeat samples and one composite raw hepatopancreas sample of Dungeness crab for use in the King County Water Quality Assessment. All King County samples were analyzed for PCBs,

[59] Note: not all COPCs were measured at each station.

PAHs, metals, volatile organic compounds (VOCs), and semivolatile organic compounds (SVOCs) (except for organochlorine and organophosphate pesticides) listed in Table A-3-4. All crab samples were collected between Slip 1 and Harbor Island.

Table A-3-4. List of compounds analyzed in raw edible meat of Dungeness crabs collected by King County in 1997

PAHs	OTHER VOCs AND SVOCs, CONTINUED	METALS AND ORGANOMETALLICS
Acenaphthene	2-Chloronaphthalene	Antimony
Acenaphthylene	2-Chlorophenol	Arsenic
Anthracene	2-Methylphenol	Cadmium
Benz(a)anthracene	2-Nitroaniline	Chromium
Benzo(a)pyrene	2-Nitrophenol	Copper
Benzo(b)fluoranthene	3,3'-Dichlorobenzidine	Lead
Benzo(g,h,i)perylene	3-Nitroaniline	Mercury
Benzo(k)fluoranthene	4,6-Dinitro-o-cresol	Nickel
Chrysene	4-Bromophenyl phenyl ether	Silver
Dibenz(a,h)anthracene	4-Chloro-3-methylphenol	Zinc
Fluoranthene	4-Chloroaniline	Tributyltin
Fluorene	4-Chlorophenyl phenyl ether	**PCBs**
Indeno(1,2,3-cd)pyrene	4-Methylphenol	Aroclor-1016/1242
2-Methylnaphthalene	4-Nitroaniline	Aroclor-1221
Naphthalene	4-Nitrophenol	Aroclor-1232
Phenanthrene	Aniline	Aroclor-1242
Pyrene	Benzidine	Aroclor-1248
PHTHALATES	Benzoic acid	Aroclor-1254
BEHP	Benzyl alcohol	Aroclor-1260
Butyl benzyl phthalate	bis(2-chloroethoxy)methane	
Diethyl phthalate	bis(2-chloroethyl)ether	
Dimethyl phthalate	bis-chloroisopropyl ether	
Di-n-butyl phthalate	Carbazole	
Di-n-octyl phthalate	Coprostanol	
OTHER VOCs AND SVOCs	Dibenzofuran	
1,2,4-Trichlorobenzene	Hexachlorobenzene	
1,2-Dichlorobenzene	Hexachlorobutadiene	
1,2-Diphenylhydrazine	Hexachlorocyclopentadiene	
1,3-Dichlorobenzene	Hexachloroethane	
1,4-Dichlorobenzene	Isophorone	
2,4,5-Trichlorophenol	Naphthalene	
2,4,6-Trichlorophenol	Nitrobenzene	
2,4-Dichlorophenol	N-Nitrosodimethylamine	
2,4-Dimethylphenol	N-Nitroso-di-n-propylamine	
2,4-Dinitrophenol	N-Nitrosodiphenylamine	
2,4-Dinitrotoluene	Pentachlorophenol	
2,6-Dinitrotoluene	Phenol	

Chemical concentrations in crab tissue from 1998 and 1997 are presented in Tables A-3-5 and A-3-6, respectively. Table A-3-6 also presents chemical concentrations measured in both edible meat and hepatopancreas tissues, and an estimate of concentrations in whole-body crab (i.e., edible meat plus hepatopancreas). Whole-body estimates were made by calculating weighted concentrations with the two tissue types, assuming edible meat was 85% of the total body weight and hepatopancreas was 15% of the total body weight. The assumed ratio of edible weight and hepatopancreas to whole body weight was based on best professional judgment.

Table A-3-5. Chemical concentrations in red rock and Dungeness crab raw edible meat collected in 1998

Chemical	Red Rock[a] (mg/kg ww)	Dungeness[b] (mg/kg ww)	Combined Red Rock and Dungeness[b] (mg/kg ww)
Mercury	0.110	0.070	0.070
TBT	<0.002	<0.002	<0.002
Total PCBs	0.164	0.080	0.060

Source: Environmental Solutions Group (1999)

[a] Maximum concentration reported in two composite samples

[b] One composite sample was collected and analyzed

Of the 84 chemicals analyzed in both studies, only 11 were detected in both edible crabmeat and hepatopancreas, including TBT, PCBs, and nine metals (arsenic, cadmium, chromium, copper, lead, mercury, nickel, silver, and zinc). Benzyl alcohol was detected in hepatopancreas tissue only. Weighted whole-body concentrations and hepatopancreas concentrations of the 11 detected chemicals presented in Table A-3-6 were used for risk characterization in Section A.7.1.2 because these concentrations incorporate hepatopancreas data and are higher than those measured in 1998. The uncertainty analysis (Section A.7.1.2) discusses chemicals for which toxicological data were available, but that were not measured in crab tissue from the LDW.

Table A-3-6. Chemical concentrations in Dungeness crab raw edible meat and hepatopancreas collected in 1997

Chemical	Edible Meat[a] (mg/kg ww)	Hepatopancreas[b] (mg/kg ww)	Estimated Whole Body[c] (mg/kg ww)
Metals			
Arsenic	12.5	6.98	11.7
Cadmium	0.022	0.122	0.037
Chromium	0.160	0.083	0.148
Copper	15.8	42.9	19.9
Lead	0.244	0.182	0.235
Mercury	0.111	0.672	0.195
Nickel	0.121	0.24	0.139
Silver	0.187	0.501	0.234
Zinc	39.1	19.1	36.1

Chemical	Edible Meat[a] (mg/kg ww)	Hepatopancreas[b] (mg/kg ww)	Estimated Whole Body[c] (mg/kg ww)
Organic Compounds			
Benzyl alcohol	nd (0.027)	0.085	nc
PAHs	nd (0.011-0.043)	nd (0.016-0.064)	nc
Phthalates	nd (0.011-0.027)	nd (0.016-0.04)	nc
SVOCs	nd (0.011-0.11[d])	nd (0.016-0.16[e])	nc
TBT	0.0819	0.0592	0.078
Total PCBs	0.177	1.65	0.398
VOCs	nd (0.011-0.11)	nd (0.016-0.16)	nc

Source: King County (1999a)

nd – not detected (detection limits in parentheses)

nc – not calculated

[a] Maximum concentration reported in two composite samples.

[b] One composite sample was collected and analyzed.

[c] Whole body concentration estimated using edible meat and hepatopancreas data; weighted concentration calculated as 85% of maximum edible meat concentration plus 15% of hepatopancreas concentration.

[d] With the exception of benzidine with a detection limit of 0.64 mg/kg ww.

[e] With the exception of benzidine with a detection limit of 0.96 mg/kg ww.

A.3.1.2.2 Exposure of benthic invertebrates to TBT

In this section, potential exposure of benthic invertebrates to TBT is assessed using a tissue residue approach, as previously discussed in Section A.3.1. Available tissue data for infaunal benthic invertebrates include four composite amphipod samples collected in the vicinity of Kellogg Island and analyzed for 125 chemicals. Of the 125 chemicals analyzed, 21 were detected in at least one of the four samples, including TBT.[60]

Measured TBT concentrations in amphipods collected in the LDW were 0.032 and 0.031 mg/kg ww in the two composite samples collected nearest Kellogg Island and 0.036 and 0.018 mg/kg ww for the two samples further west of Kellogg Island (referred to as West Marginal Way samples). Because TBT sediment concentrations near Kellogg Island are not representative of the range of TBT concentrations in bulk sediment from the entire LDW, a bioaccumulation factor was calculated to estimate the range of concentrations potentially present in amphipods elsewhere in the LDW. Bioaccumulation factors are generally expressed as either the tissue to sediment concentration ratio (BAF; Equation 3-1), or the biota-sediment accumulation factor (BSAF; Equation 3-2) for those chemicals more closely associated with lipid in tissue and organic carbon in sediment, as follows:

$$BAF = \frac{\text{Biota (mg/kg dry weight)}}{\text{Sediment (mg/kg dry weight)}} \qquad \textit{Equation 3-1}$$

[60] In addition to TBT, antimony, arsenic, cadmium, chromium, copper, lead, mercury, nickel, silver, zinc, Aroclor 1248, Aroclor 1254, Aroclor 1260, total PCBs, BEHP, phenol, fluoranthene, pyrene, dibutyltin, and monobutyltin were detected in at least one amphipod tissue sample.

$$BSAF = \frac{\text{Biota (mg/kg lipid)}}{\text{Sediment (mg/kg OC)}}$$ *Equation 3-2*

For TBT, percent lipid in the organism does not appear to control TBT bioaccumulation; the sediment organic-carbon content appears to be more relevant (Meador 2000). Therefore, a modified bioaccumulation factor (Equation 3-3), using a wet weight tissue concentration and a sediment concentration expressed on an organic carbon-normalized basis, was calculated as follows:

$$\text{Modified BAF for TBT} = \frac{\text{Biota (mg/kg wet weight)}}{\text{Sediment (mg/kg OC)}}$$ *Equation 3-3*

Table A-3-7 presents the modified BAF for TBT using co-located amphipod and sediment data. The sediment and tissue concentrations were very similar at the two locations, resulting in similar BAFs. Meador (2000) reported dry-weight BAFs for field-collected sediments (calculated using Equation 3-1) in the range of 3–100. Similar calculations using the data from Table A-3-7 yield dry-weight BAFs of 38 and 12 for Kellogg Island and West Marginal Way, respectively. Thus, data from this site are consistent with BAFs reported in the literature.

Table A-3-7. Modified bioaccumulation factor for TBT calculated with co-located amphipod and sediment data[a]

Location	Biota (mg/kg ww)	OC-normalized Sediment Conc.[a] (mg/kg OC)	Modified BAF[b]
TBT			
Kellogg Island	0.032	0.32	0.10
West Marginal Way	0.027	0.41	0.066

[a] Sediment values are means of three Kellogg Island samples (each replicated 3 times) and three West Marginal Way samples (not replicated).

[b] Calculated using Equation 3-3

A range of amphipod concentrations elsewhere in the LDW was estimated using two modified BAFs (0.066 and 0.10), along with the following sediment concentrations: minimum, median, and maximum concentration of TBT (Table A-3-8). Tissue concentrations measured near Kellogg Island and West Marginal Way (0.018 - 0.036 mg/kg ww) were two orders of magnitude lower than the TBT tissue concentration estimated from the maximum sediment concentration (Table A-3-8).

Table A-3-8. Range of sediment concentrations used to estimate amphipod tissue concentrations and the estimated amphipod tissue concentrations

Sediment Statistic	Measured TBT Conc. in Sediment (mg/kg OC)	Estimated TBT in Tissue (mg/kg ww)
Minimum concentration	0.053	0.0035 – 0.0053
Median concentration [a]	2.0	0.13 – 0.2
Maximum concentration	36	2.4 – 3.6

[a] Organic carbon concentrations were not available for 6 of the 135 samples that were analyzed for TBT, so these data were not included in the calculations.

The risk characterization (Section A.7.1.1.1) quantitatively assesses risks using measured TBT tissue concentrations (lower of maximum or 95% UCL on the mean concentration). The maximum measured TBT tissue concentration (i.e., 0.036 mg/kg ww) was lower than the 95% UCL on the mean (i.e., 0.040 mg/kg ww), and thus was used in the risk characterization. In addition, risks based on the range of TBT concentrations estimated in tissue were also assessed in the risk characterization. The greater uncertainty associated with this assessment is discussed in the uncertainty section (Section A.7.1.2).

A.3.2 Effects Assessment

Section A.3.1 characterized the potential exposure of benthic invertebrates to COPCs and also compared COPC concentrations to effects-based sediment standards or guidelines (SQS/SL and CSL/ML). The SMS standards and DMMP guidelines were presented in Section A.3.1 to provide a frame of reference for discussing chemical concentrations. The effects concentrations used in the risk characterization (Section A.7.1.1) include not only the standards or guidelines, but also the AETs for multiple endpoints on which these standards and guidelines are based. Section A.3.2.1 provides additional information regarding these standards and guidelines relative to the AETs.

Because AETs were determined with sediment samples containing chemical mixtures, attributing toxicity to a single chemical can be difficult. Therefore, the SMS regulations provide for a site-specific verification of toxicity using sediment toxicity tests and benthic community characterizations. Sections A.3.2.2 and A.3.2.3 describe the limited available site-specific biological and toxicological data collected to assess the potential effects on benthic invertebrates in the LDW. While these data provide an indication of toxicity and adverse community effects at specific locations within the LDW, they do not provide cause-and-effect relationships between specific chemical concentrations and these observed effects due in part to the very limited dataset and the mixture of chemicals in the samples.

Tissue concentrations associated with effects on crabs (multiple COPCs) and benthic invertebrates (TBT) are presented in Sections A.3.2.4 and A.3.2.5, respectively.

A.3.2.1 AETs

The SQS and CSL standards were derived in 1991 when the Washington Department of Ecology adopted the SMS. These standards were based on AETs developed for the Puget Sound Estuary Program (PTI 1988). The methods used to calculate the AETs are described in PTI (1988) and Gries and Waldow (1996). AETs were empirically derived using data from field-collected sediment samples that contained diverse chemical mixtures analyzed simultaneously for chemistry and toxicity. The data used to derive the 1988 AETs were collected between March 1982 and September 1986. An AET is the highest "no effect" chemical-specific sediment concentration above which a significant adverse biological effect always occurred among the several hundred samples used for its derivation. During AET development, synoptic sediment samples were placed into two groups based on whether statistically significant adverse biological effects, as compared to a reference sample, occurred in the specific toxicity test conducted. Outliers and statistically inconclusive data points were excluded. This process was repeated for multiple chemicals and endpoints.

AETs for four endpoints[61] (amphipod survival, abnormal development of oyster larvae, benthic community, and Microtox®) were developed for 47 chemicals (Table A-3-9). The lowest AET for each chemical was identified as the SQS; the second lowest was identified as the CSL. Section 130(6) of the SMS rule requires a periodic review and revision, if necessary, of the rule, including the SQS and CSL. The first extensive review was conducted in 1996, using data collected through 1994 (Gries and Waldow 1996). Table A-3-9 shows the four original AETs for each chemical (i.e., 1998 amphipod and benthic, 1986 Microtox® and oyster) and the two additional AET sets calculated during the 1996 review (i.e., 1994 amphipod and echinoderm). The latter AETs were never formally adopted as part of the SMS rule.

Additional review of the SQS and CSL continued as part of the SMS rule revision that began in 1997. AETs for the *Neanthes* and mussel endpoints were developed and the amphipod and echinoderm AETs were updated during this review. The Regulatory Work Group, a panel of 15 experts with extensive technical and policy expertise in sediment quality issues, recommended that the new mussel abnormality AETs replace the 1986 oyster abnormality AETs and that the new *Neanthes* growth AETs replace the 1986 Microtox® AETs, unless the predictive ability of the suite of AETs would be substantially diminished by doing so (Gries 1999). However, these recommendations were ultimately not implemented when the SMS rule revision was halted at the end of 1999 (Fitzsimmons 1999). Since the revised AETs were never formally published and some technical issues regarding their calculation remain unresolved, they are not discussed further here. The new mussel and *Neanthes* AETs are not shown in Table A-3-9 because of the unresolved issues.

---

[61] The specific tests associated with each of these endpoints are described in greater detail in the SMS rule (WAC 173-204).

Table A-3-9. Puget Sound Apparent Effects Thresholds (AETs)

Chemical Group/ Chemical of Concern	1994 Amphipod AET	1988 Amphipod AET	1994 Echinoderm AET	1988 Benthic AET	1986 Microtox® AET	1986 Oyster AET
Metals (mg/kg dw)						
Antimony	200	200	9.3	150	na	na
Arsenic	450	93	130	57	700	700
Cadmium	14	6.7	2.7	5.1	9.6	9.6
Chromium	>1,100	270	>96	260	na	na
Copper	1,300	1,300	390	530	390	390
Lead	1,200	660	430	450	530	660
Mercury	2.3	2.1	1.4	2.1	0.41	0.59
Nickel	>370	>140	110	>140	na	na
Silver	6.1	6.1	8.4	>6.1	>0.56	0.56
Zinc	3,800	960	460	410	1,600	1,600
Organic compounds (µg/kg dw)						
Low molecular weight polycyclic hydrocarbons (PAHs)						
LPAH	29,000	24,000	1,200	13,000	5,200	5,200
2-Methylnaphthalene	1,900	1,900	64	1,400	670	670
Acenaphthene	2,000	2,000	130	730	500	500
Acenaphthylene	1,300	1,300	71	1,300	>560	>560
Anthracene	13,000	13,000	280	4,400	960	960
Fluorene	3,600	3,600	120	1,000	540	540
Naphthalene	2,400	2,400	230	2,700	2,100	2,100
Phenanthrene	21,000	6,900	660	5,400	1,500	1,500
High molecular weight PAHs						
HPAH	69,000	69,000	7,900	69,000	12,000	17,000
Benz(a)anthracene	5,100	5,100	960	5,100	1,300	1,600
Benzo(a)pyrene	3,500	3,000	1,100	3,600	1,600	1,600
Benzo(g,h,i)perylene	3,200	1,400	920	2,600	670	720
Benzofluoranthenes	9,100	7,800	1,800	9,900	3,200	3,600
Chrysene	21,000	9,200	950	9,200	1,400	2,800
Dibenz(a,h)anthracene	1,900	540	240	970	230	230
Fluoranthene	30,000	30,000	1,300	24,000	1,700	2,500
Indeno(1,2,3-c,d)pyrene	4,400	1,800	760	2,600	600	690
Pyrene	16,000	16,000	2,400	16,000	2,600	3,300
Chlorinated organic compounds						
1,2,4-trichlorobenzene	51	51	>4.8	na	31	64
1,2-dichlorobenzene	>110	>110	na	50	35	50
1,3-dichlorobenzene	>170	>170	>4.4	>170	>170	>170
1,4-dichlorobenzene	120	120	na	110	110	>120
Hexachlorobenzene	130	130	na	22	70	230

Chemical Group/ Chemical of Concern	1994 Amphipod AET	1988 Amphipod AET	1994 Echinoderm AET	1988 Benthic AET	1986 Microtox® AET	1986 Oyster AET
Phthalates						
BEHP	>8,300	>3,100	1,700	1,300	1,900	1,900
Butyl benzyl phthalate	970	900	200	900	63	>470
Di-n-butyl phthalate	1,400	1,400	>31	>5,100	1,400	1,400
Di-n-octyl phthalate	>2,100	>2,100	>98	6,200	na	>420
Diethylphthalate	>1,200	>1,200	>62	200	>48	>73
Dimethylphthalate	>1,400	>1,400	85	>1,400	71	160
Phenols						
2-methyl phenol	77	63	63	72	>72	63
2,4-dimethyl phenol	77	72	55	210	29	29
4-methyl phenol	3,600	3,600	670	1,800	670	670
Pentachlorophenol	400	360	150	690	>140	>140
Phenol	1,200	1,200	420	1,200	1,200	420
Miscellaneous Extractables						
Benzyl alcohol	73	870	>12	870	57	73
Benzoic acid	760	760	>31	650	650	650
Dibenzofuran	1,700	1,700	110	700	540	540
Hexachlorobutadiene	180	180	1.3	11	120	270
Hexachloroethane	140	na	na	na	na	na
N-nitrosodiphenylamine	48	48	>25	28	40	130
Volatile organics						
Ethylbenzene	50	>50	4.0	10	33	37
Tetrachloroethene	>210	>210	1.0	57	140	140
Xylene, total	160	>160	>21	40	100	120
Pesticides and PCBs						
Aldrin	9.5	na	9.5	na	na	na
Chlordane	2.8	na	>4.5	na	na	na
Dieldrin	3.5	na	1.9	na	na	na
Heptachlor	1.5	na	2.0	na	na	na
p,p'-DDD	63	43	28	16	na	na
p,p'-DDE	62	15	9.3	9.0	na	na
p,p'-DDT	>270	>270	12	34	na	>6
Total DDT	24	na	37	na	na	na
Total PCBs	3,100	3,100	450	1,000	130	1,100

Sources: 1986 and 1988 AETs from PTI (1988); 1994 AETs from Gries and Waldow (1996)

">" symbol indicates there is no "hit" sample with a greater concentration than the reported AET, which should be thought of as a minimum

na - not available

The SLs and MLs used by DMMP are based on the same 1988 AETs used to develop the SMS (PTI 1988). The highest of the four AETs established the ML for a given chemical. The SL is generally equal to the lowest dry weight AET (Gries 1997).

A.3.2.2 Site-specific toxicity tests

Although sediment chemistry is an important component for evaluation of risks to benthic invertebrates, synoptic toxicity tests and benthic community analysis usually strengthen the assessment (Long and Chapman 1985). With respect to site-specific toxicity tests, 12 studies have been conducted in the LDW within the last 10 years[62] that included sediment toxicity tests on a total of 54 samples (Table A-3-10). However, only 2 of these 12 studies (shown in bold in Table A-3-10) were conducted on surface sediments (0-15 cm) that remained in place (10 samples); the remainder were conducted for dredged material characterization studies that tested sediments from the 0-4 ft (or deeper) horizons. The dredged material characterization studies listed in Table A-3-10 were not appropriate for evaluating the relationship between surface sediment chemistry and toxicity for the purposes of the benthic invertebrate effects assessment. These sediments were located below the biologically active zone and do not have equivalent ecological functions compared to sediment from the 0-15 cm horizon.

The summary presented below focuses on the two studies that included surface sediment toxicity tests (Ecology 2000; King County 2000). Map A-3-7 shows collection locations for samples analyzed for sediment toxicity in these studies. The samples analyzed for the Duwamish/Diagonal cleanup study (King County 2000) were collected in an area with moderately high chemical concentrations. Five of the seven locations sampled were within an area proposed for cleanup by King County (2000), in part, due to elevated concentrations of BEHP. The three locations sampled by Ecology (2000) were collected for reconnaissance purposes and were not targeted on a particular contaminant source.

Seven sediment samples collected for the Duwamish/Diagonal cleanup study (King County 2000) were analyzed for toxicity using three standard SMS confirmatory tests (amphipod mortality, echinoderm effective[63] mortality, and *Neanthes* growth). One of seven samples (L9443-7) failed the biological effects standard of the SMS at the SQS level for both the echinoderm embryo survival/development and the polychaete *Neanthes* growth endpoints (Table A-3-11). The results for the *Neanthes* and embryo tests for Sample L9443-7 were similar to the results from several other samples tested concurrently. The control sample to which the results from Sample L9443-7 were compared had a higher *Neanthes* growth rate (0.77 vs. 0.60 mg/day) and lower effective echinoderm embryo mortality (15% vs. 29%) compared to the reference sample to which all other test sediments were compared.[64]

---

[62] Data older than 10 years are considered unsuitable for characterizing surface sediment. At the beginning of this project, the samples collected in 1990 and 1991 were not older than 10 years. These older events, which are now older than 10 years, are included in Table A-3-10 to remain consistent with the list of suitable data sets developed at the beginning of the project.

[63] Combined mortality and abnormal development

[64] This sample was not compared to the other reference samples because they were not suitable grain size matches. The percent fines for sample L9443-7 (7.9%) was much lower than the remaining

Table A-3-10. Sediment toxicity datasets that met project data quality objectives

Report Title	Year Conducted	Citation	Number of Samples [a]
Sediment Quality in Puget Sound	**1998**	**Ecology (2000)**	**3**
Duwamish/Diagonal Cleanup Study-Draft	**1996**	**King County (2000)**	**7**
Sediment sampling and analysis - James Hardie Gypsum Inc.	1998-1999	Spearman (1999)	7
Dredge Material Characterization Hurlen Construction Company & Boyer Alaska Barge Lines Berthing Area	1998	Hart Crowser (1998)	4
Proposed Dredging of Slip No. 4, Duwamish River, Seattle, WA	1995	PTI (1996)	4
1996 USACE Duwamish O&M	1996	Striplin Environmental (1996a)	3
Lone Star Northwest and James Hardie Gypsum-Kaiser Dock upgrade	1995	Hartman Associates (1995)	4
Lonestar Northwest - West Terminal USACE – Seattle	1992	Hartman Associates (1992)	1
South Park Marina maintenance dredging, 1991	1991	Spearman (1991a)	2
Sediment Sampling Analysis Brown and Morton Properties Duwamish Waterway	1991	Spearman (1991b)	1
PSDDA Bioassays for Duwamish Channel Sediments (O&M)	1991	SAIC (1992)	14
Duwamish River Maintenance Dredge, Phase 1	1990	PTI (1990)	4

Note: **Studies shown in bold** characterized surface sediments; all other studies were dredged material characterizations and are not discussed further.

[a] Subsurface sediment samples from above studies (all except two studies in bold) were collected for dredged material characterization studies. The tested sediment has been removed.

samples (49.5% to 91.3%) and in-batch reference sediment (54.5%). Therefore, the SMS comparison was performed on the West Beach control sediment, which had <10 % fines.

Table A-3-11. Summary of site-specific sediment toxicity test results for surface sediment samples collected at Duwamish/Diagonal CSO/SD site

Location	Sample	Echinoderm Embryo - Effective % Mortality	*Neanthes*-Growth Rate (mg/day)	Amphipod - % Mortality
West Beach	Control	15	0.77	1.0
Carr Inlet	Reference	29	0.60	8.0
DUD200	L9443-1	32	0.60	13
DUD201	L9443-2	35	0.55	21
DUD202	L9443-3	35	0.62	18
DUD203	L9443-4	33	0.59	22
DUD204	L9443-5	17	0.51	26
DUD205	L9443-6	16	0.54	19
DUD206	L9443-7[a]	34 (SQS)	0.52 (SQS)	4.0

Source: King County (2000)

Note: SQS indicates biological effects standard specified in SMS were exceeded. Samples without any such designation are considered non-toxic with respect to SMS standards.

[a] Test results for this sample were statistically compared to control sample results rather than reference sample results because neither reference sample was a suitable grain size match.

Three sediment samples were analyzed for toxicity by Ecology (2000) using four different tests: amphipod toxicity (*Ampelisca abdita*) in bulk sediment, sea urchin fertilization (*Strongylocentrotus purpuratus*) in pore water, Microtox® bioluminescence in an organic extract from bulk sediment, and human reporter gene system (RGS; Cytochrome P450) response to an organic extract from bulk sediment. The data from these tests are shown in Table A-3-12. Based on information presented in the summary report (Ecology 2000), these three samples were considered non-toxic based on results from the three toxicity tests that included controls (i.e., all but the RGS test). The RGS test is generally used more as an estimate of exposure than effects. Correlation between the gene response, in benzo(a)pyrene equivalents, and the presence of toxic chemicals has been demonstrated (Anderson et al. 1995), but the assay itself does not measure toxicity. Thus, based on the available data, only one of the ten sediment samples analyzed by King County (2000) and Ecology (2000) was considered toxic to test organisms based on SQS exceedances.

Table A-3-12. Summary of site-specific sediment toxicity test results for surface sediment samples collected by Ecology (2000)

Sample	Amphipod Survival (as % of control)	Mean Urchin Fertilization in 100% Porewater (as % of control)	Microtox® EC50 (mg/mL)	Cytochrome P-450 RGS (as µg B[a]P/g)
203	103	98	3.20 [a]	96.9 [b]
204	92	103	3.33 [a]	77.0 [b]
205	101	94	3.57 [a]	46.9 [b]

Source: Ecology (2000)

[a] EC50s are well above 0.51 mg/mL, which was determined as the 80% lower prediction limit. Result is considered a negative response.

[b] Value is > 37.1 benzo[a]pyrene equivalents (µg/g sediment) determined as the 90% upper prediction limit. Result is considered a positive response.

Each surface sediment sample tested for toxicity to benthic invertebrates was also analyzed for chemistry. Analysis of the toxicity and chemistry results in combination is provided in the uncertainty assessment (Section A.7.1.2).

A.3.2.3 Site-specific benthic community data

Five benthic community characterization studies have been conducted in the LDW (Table A-3-13). Map A-3-7 shows collection locations for samples analyzed for benthic community characteristics. These studies are discussed further below.

Table A-3-13. Benthic macroinvertebrate datasets collected within past 10 years

Report Title	Year Conducted	Citation	Number of Samples
Sediment Quality in Puget Sound	1998	Ecology (2000)	3
King County Combined Sewer Overflow Water Quality Assessment for the Duwamish River and Elliott Bay - Benthic Task	1997	King County (1999a)	6
Duwamish Coastal America Restoration and Reference Sites: Results from 1997 Monitoring studies	1997	Cordell et al. (1999)	21
Duwamish Coastal America Restoration and Reference Sites: Results from 1996 Monitoring studies	1996	Cordell et al. (1997)	21
Duwamish Coastal America Restoration and Reference Sites: Results from 1995 Monitoring studies	1995	Cordell et al. (1996)	6

Six samples analyzed for the King County Water Quality Assessment (1999a) were collected near Duwamish/Diagonal CSO/SD (three samples) and Kellogg Island (three samples identified as reference samples by the authors). The Duwamish/Diagonal area has been the focus of environmental investigations since 1994. The results for these samples are presented in Table A-3-14. The SQS for benthic invertebrate community samples include tests of significant difference ($p<0.05$) and a 50% or greater reduction in the mean abundance of polychaetes, crustaceans, and/or mollusks relative to reference samples to determine if an area is "impaired." Results from this survey were presented and discussed by Striplin Environmental (1998). All

three Duwamish/Diagonal stations failed the SQS biological effects criteria for one or more abundance endpoints, and were thus designated as impaired.

Table A-3-14. Summary of benthic macroinvertebrate community results [a] for King County Water Quality Assessment survey (September 1997)

Sample [b]	Total Abundance	Polychaete Abundance	Crustacean Abundance	Mollusk Abundance
Pair 1				
DD-1	260	**78.7** [c]	**3.3** [c]	**0.3** [c]
KI-4	5,445	3,600	1,674	32.0
Pair 2				
DD-3	1,059	887	39.3	**109** [c]
KI-2	555	244	65.7	222
Pair 3				
DD-5	800	**320** [c]	92.3	350
KI-1	1,357	1,045	55.0	252

[a] Results for each sample are mean values (number of animals/0.1 m^2) from the analysis of three replicates prepared in the field with a 1.0-mm sieve.

[b] Duwamish/Diagonal (DD) and Kellogg Island (KI) reference station pairs shown in table were determined by Striplin Environmental (1998) based on similarities between water depth, percent fines, and sediment total organic carbon.

[c] Mean Duwamish-Diagonal abundances in bold represent exceedances of SQS standards. Statistical comparisons in abundance were made within each pair identified in table.

The King County results were also compared to reference ranges for benthic communities developed for Ecology (Striplin 1996b).[65] Values at Station DD-1 were depressed below the reference range for 12 of 14 benthic community indices (data not shown), indicating the site was severely impacted. Station DD-3 was the next most impacted; it was below the reference range for 9 of 14 benthic community indices. This station had the highest total abundance of the three Duwamish-Diagonal stations, but was strongly dominated by the polychaetes *Aphelochaeta* spp. and *Capitella capitata*, both of which are thought to be indicators of organic enrichment. Station DD-5 appeared to be the least impacted. It was depressed below the reference range for 7 of 14 benthic community indices. This station had fewer "indicator" species and had a greater diversity of benthic infauna compared to the other two Duwamish/Diagonal stations. Although the Kellogg Island stations were intended to represent local reference conditions, values from each station were depressed below reference ranges, suggesting these stations may not have been suitable references. Values at Stations KI-

[65] Reference ranges were calculated by habitat category (water depth and grain size) within Puget Sound. Each range represents the mean plus or minus two standard deviations for all samples at which no SQS exceedances were found. Ranges were calculated for the following benthic community endpoints: total abundance, total taxa, crustacean abundance, crustacean taxa, amphipod abundance, amphipod taxa, polychaete abundance, polychaete taxa, mollusk abundance, mollusk taxa, Shannon-Wiener Diversity (H'), Pielou's Evenness Index (J'), Infaunal Trophic Index (ITI), Swartz's Dominance Index (SDI).

4, KI-1, and KI-2 were below reference ranges for 7, 6, and 3 of the 14 metrics, respectively.

Ecology (2000) evaluated benthic community characteristics at three sites in the LDW (Map A-3-7, Table A-3-15); this was the only study to include the synoptic analysis of benthic community and sediment chemistry. However, matched reference samples were not collected for this survey. Ecology (2000) found all three samples to have a low Swartz's Dominance Index (SDI < 10). Sample 203 had the highest total abundance and species richness. Pollution-tolerant species *Aphelochaeta* spp., *Scoletoma luti*, and *Nutricola lordi* were the most abundant taxa found in all three samples. Ecology (2000) concluded that Samples 204 and 205 displayed "pollution-induced degradation." Evenness and Swartz's Dominance Index were lower for each of the three samples than the range of reference values compiled by Striplin (1996b). The benthic community characteristics at these stations, however, were consistent with those typically found in areas of organic enrichment, and are not necessarily indicative of chemical contamination (Pearson and Rosenberg 1978).

Table A-3-15. Summary of benthic macroinvertebrate community results for Ecology sediment quality of Central Puget Sound survey[a]

Sample	Total Abundance	Taxa Richness	Evenness	Swartz's Dominance Index	Annelid Abundance	Arthropod Abundance	Mollusca Abundance	Echinoderm Abundance
203	3,764	94	0.426	3.0	2,970	94	688	0
204	1,155	52	0.373	2.0	1,002	31	117	1
205	1,561	65	0.454	3.0	1,314	17	226	1

[a] June 1998

Note: Each sample is a single sample (not replicated) prepared in the field with a 1.0-mm sieve. Abundance values are number of animals/0.1-m^2.

Intertidal restoration sites within the LDW were sampled by Cordell et al. (1996, 1997, 1999) to determine colonization success of benthic invertebrates in created and restored habitats (Map A-3-7). Some of the created habitats are becoming colonized by ecologically important benthic invertebrates (Cordell et al. 1999). Data from these monitoring studies are not discussed here because synoptic chemistry data were not available, and the sampling sites do not reflect current sediment contamination in the LDW because they have been restored or created.

Analysis of the benthic community and any corresponding chemistry results will be provided in the uncertainty assessment (Section A.7.1.2).

A.3.2.4 Effects data for crabs

This section presents chemical tissue residue data associated with effects on crab survival, growth, and reproduction. Effects data related to crab tissue concentrations were identified by searching the following databases: Environmental Residue Effects Database (ERED; ACOE 2002); Jarvinen and Ankley (1999) tissue residue database;

URS database (Hoffman 2001); and BIOSIS previews. The literature search focused on crab studies, but included other decapod species if crab data were not available for individual chemicals detected in LDW crab tissue (see Tables A-3-5 and A-3-6 for detected chemicals). BIOSIS was searched for chemicals detected in LDW crab tissue only, whereas the other databases were searched for all chemicals with tissue effects data. Original sources of toxicity data were obtained and reviewed to verify effects data summarized in the databases as well as the suitability of the studies. To be acceptable, it was necessary for studies to include adequate controls and statistical evaluation of effects.

Tissue-based toxicological data for crabs were found for chromium, mercury, PCBs, TBT, chlordecone, DDT, methoxychlor, Mirex, 1,2,3-trichlorobenzene, and 1,2,3,4-tetrachlorobenzene. This list includes four of the 11 chemicals detected in LDW crab tissue (chromium, mercury, PCBs, and TBT; Tables A-3-5 and A-3-6). For the remaining seven chemicals detected in LDW crabs, the databases were searched for other decapod toxicity data; shrimp or crayfish data were found for four of these remaining chemicals (arsenic, cadmium, copper, and zinc). Table A-3-16 summarizes the toxicity data available for these eight chemicals for which either crab or other decapod toxicity data were available. Although a crab study was found for mercury, the exposure time was only 32 hours, so a study with lobster was also included in Table A-3-16. The remaining three chemicals detected in LDW crab tissue, but for which no toxicity data were available (lead, nickel, and silver), are discussed in the uncertainty assessment (Section A.7.1.2).

No studies with reproductive endpoints were identified; all studies addressed either growth or survival. NOEC and LOEC concentrations were available for cadmium, chromium, mercury and zinc, whereas only NOECs were available for arsenic, copper, PCBs, and TBT. These effects data are compared to available crab tissue data collected in the LDW in the risk characterization (Section A.7.1.1).

A.3.2.5 Tissue-based toxicity data for TBT

Effects on survival, growth, and reproduction of benthic invertebrates associated with tissue residues of TBT are discussed in this section. In the risk characterization (Section A.7.1.1.2), these data are compared to measured amphipod tissue data as well as the estimated range of TBT concentrations to evaluate risks to benthic invertebrates from TBT.

Potential effects from exposure to sediment-associated TBT have been discussed in detail in EPA (1999) and Meador (2000). Both documents recommend a critical tissue residue concentration of 3 mg/kg dw (0.6 mg/kg ww, assuming 20% solids). This value was derived using a weight of evidence approach in EPA (1999). Two of the approaches used in developing the critical tissue residue concentration were an

Table A-3-16. Effects associated with body burdens in crab and other decapods for chemicals detected in LDW crab tissue

Chemical	Chemical Form Administered	Species	Exposure Route and Duration	Tissue Type Analyzed	NOEC Conc (mg/kg ww)	LOEC Conc (mg/kg ww)	Endpoint	Reference
Arsenic	Sodium arsenate	Juvenile grass shrimp *(Palaemonetes pugio)*	10 µg/L in water for 28 days	Whole body	1.15[a]		Growth	Lindsay and Sanders 1990
Cadmium	Cadmium chloride	Shore crab (*Carcinus maenas*)	1 mg/L in water for 40 days	Muscle	4.9[a,b]	9.5[a,b,c]	Survival	Jennings and Rainbow 1979
Chromium	Potassium dichromate	Juvenile (2nd instar) sand crab (*Portunus pelagicus*)	0.1 mg/L in water for 30 days	Whole body	1		Growth	Mortimer and Miller 1994
	Potassium dichromate	Juvenile (2nd instar) sand crab (*Portunus pelagicus*)	0.3 mg/L in water for 30 days	Whole body		3.2	Growth	Mortimer and Miller 1994
Copper	Copper sulfate	Adult crayfish (*Orconectes rusticus*)	3 mg/L in water for 2 days	Claws	34[a,d,e]		Survival	Evans 1980
Mercury	Mercuric chloride	Adult male shore crab (*Eriocheir sinensis*)	1 mg/L in water for 32 hours	Hepatopancreas		1.0[e,f]	Survival	Bianchini and Gilles 1996
	Mercuric chloride	Adult Norway lobster (*Nephrops norvegicus*)	164 mg/kg ww in diet for 50 days	Hepatopancreas	0.99[a]		Survival	Canli and Furness 1995
	Mercuric chloride	Adult Norway lobster (*Nephrops norvegicus*)	10 µg/L in water for 30 d	Hepatopancreas	1.43[a]		Survival	Canli and Furness 1995
Zinc	Zinc sulfate	Adult crayfish (*Oronectes virilis*)	12.2 µg/L in water for 14 days	Hepatopancreas	42.5[a]		Survival	Mirenda 1986
	Zinc sulfate	Adult crayfish (*Oronectes virilis*)	26.8 µg/L in water for 14 days	Hepatopancreas		85.6[a, g]	Survival	Mirenda 1986
PCBs	Aroclor 1254	Juvenile blue crab (*Callinectes sapidus*)	3.5 to 4.2 µg/L in water for 20 days	Whole body	23		Survival	Duke et al. 1970
TBT	Tributyltin chloride	Juvenile blue crab (*Callinectes sapidus*)	1.89 mg/kg ww in diet for 16 days	Whole body	0.12		Growth	Rice et al. 1989

[a] Converted from dry weight to wet weight using a moisture content of 80% (Jarvinen and Ankley 1999; 80% is also the average moisture content of two crab samples collected by King County in 1997).

[b] Concentration is lowest of five tissue types (midgut gland, gills, exoskeleton, muscle tissue, and remaining tissue).

[c] Exposed for an average of 12.3 days, at which time 50% mortality occurred.

[d] Concentration is lowest of three tissue types (claws, thorax, and abdomen).

[e] Due to short exposure time, full equilibrium between water and tissue may not have been reached.

[f] Concentration is lowest of three crab species tested (*Carcinus maenas*, *Eriocheir sinensis*, and *Cancer pagurus*).

[g] Survival reduced 22%.

evaluation of sublethal effects data, including sterilization due to imposex (Table A-3-17), and critical body residue data associated with growth effects and bioconcentration factors for six species including one trout species, two polychaete species, two oyster species, and a mussel species (EPA 1999; Meador 2000).

Table A-3-17. TBT sublethal effects tissue data from EPA (1999)[a]

Species	Endpoint	TBT LOEC (µg/g dw)	Reference
Snail (*Ocinebrina aciculate*)	sterilization due to imposex	1.1	Oehlmann et al. 1996
Dogwhelk (*Nucella lapillus*)	sterilization due to imposex	1.39	Gibbs et al. 1988
Dogwhelk (*Nucella lapillus*)	sterilization due to imposex	2.65	Bailey and Davies 1991
Dogwhelk (*Nucella lapillus*)	sterilization due to imposex	3.39	Bryan et al. 1987
Pacific oysters (*Crassostrea gigas*)	reduced condition index relative to control stations	3.75	Davies et al. 1988
Blue mussel (*Mytilus edulis*)	reduced growth rate	5.44	Widdows and Page 1993
Blue mussel (*Mytilus edulis*)	growth rate inhibition	6.0	Salazar and Salazar 1998
Polychaete worm (*Neanthes arenaceodentata*)	reduced reproduction	6.27	Moore et al. 1991
Dogwhelk (*Nucella lapillus*)	sterilization due to imposex	8.52	Bryan et al. 1987

[a] Based on EVS Solutions (1999)

TBT exposure is also associated with two sublethal effects specific to a small group of species, bivalve shell thickening in oysters, and induction of imposex or intersex[66] in gastropod snails. Shell thickening in oysters was not considered as a viable endpoint in the development of the tissue residue concentration discussed above due to the lack of habitat for oysters within the West Waterway of the Duwamish River (EPA 1999). The onset of imposex was also not considered an effect endpoint for the tissue residue concentration derivation. However, sterilization resulting from the advanced stages of imposex was included as a population-level effect. In the weight-of-evidence approach used to support the critical residue concentration of 3 mg/kg dw in EPA (1999), tissue concentrations associated with gastropod sterilization resulting from imposex were used to calculate the 10th and 50th percentiles of the sublethal effects dataset (1.33 and 3.75 mg/kg dw TBT, respectively) and a LOEC based on gastropod sterilization (1.1 mg/kg dw TBT) was used as a lower bound in deriving the critical residue value of 3 ppm dw TBT (EPA 1999).

In addition to the tissue residue concentration derived for the West Waterway (3.0 mg/kg dw TBT), LOEC and NOEC values associated with sterilization resulting from imposex were reviewed for this ERA. LOECs associated with sterilization resulting from advanced stages of imposex are presented in Table A-3-18.

[66] As stated in EPA (1999), imposex is defined as the development of male sexual characteristics in females. Intersex is characterized as any disturbance of phenotypic sex determination between gonad and genital tract (see Bauer et al. 1997).

Three species have been shown to develop imposex from exposure to TBT (Table A-3-18; *Littorina littorea, Ocinebrina aciculate, Nucella lapillus*); all of these species are members of the order Mesogastropodae or Neogastropodae. In the risk characterization, the residue concentration of 3 mg/kg dw is used for calculating risk. Due to the uncertainty surrounding the site usage of species sensitive to imposex (meso- or neogastropod species) in the LDW, an additional effects benchmark of 0.5 mg/kg dw proposed by Meador (2000) to be protective of reproductive impairment attributable to the onset of imposex was not used in the Phase 1 risk characterization; its potential usage will be discussed with the agencies in Phase 2. Further discussion of this uncertainty is presented in the uncertainty section (A.7.1.2.2).

Table A-3-18. Summary of available toxicity literature related to TBT and sterilization resulting from imposex

Species	Study Conditions	Effects Concentration (mg/kg dw)	Effect Endpoint	Reference
Periwinkle (*Littorina littorea*)	Field collected organisms	LOEC: 0.72 [a,b]	40% sterilization[c]	Oehlmann et al. 1998
Snail (*Ocinebrina aciculate*)	Field collected organisms	LOEC: 1.1	Sterilization due to imposex	Oehlmann et al. 1998
Dogwhelk (*Nucella lapillus*)	Aqueous exposure to 7-12ng TBT/L	LOEC: 1.39	100% sterilization	Gibbs et al. 1988
Periwinkle (*Littorina littorea*)	Field collected organisms	LOEC: 1.4	60% sterilization [c]	Bauer et al. 1997
Dogwhelk (*Nucella lapillus*)	Field collected organisms	LOEC: 2.65[a]	Sterilization due to imposex	Bailey and Davies 1991
Dogwhelk (*Nucella lapillus*)	Field transplanted mussels for 18 mo	LOEC: 3.39	Sterilization due to imposex	Bryan et al. 1987
Dogwhelk (*Nucella lapillus*)	Aqueous exposure to 107 ng TBT/L for 12 mo	LOEC: 8.52	Sterilization due to imposex	Bryan et al. 1987
Periwinkle (*Littorina littorea*)	Field collected organisms	NOEC: 0.3 [a,b,d]	Sterilization due to imposex	Oehlmann et al. 1998
Dogwhelk (*Nucella lapillus*)	Aqueous exposure to 2-5 ng TBT/L	NOEC: 0.61	Sterilization due to imposex	Gibbs et al. 1988

[a] Concentration calculated assuming a moisture content of 80 percent.

[b] Value estimated from non-linear regression in figure in original paper.

[c] Refers to the percent of sampled organisms that were found to be sterile due to imposex or intersex.

[d] No effects concentration was taken from background; therefore it may not represent the highest NOEC.

A.3.3 Summary of Benthic Invertebrate Assessment

A.3.3.1 Exposure assessment

Exposure to the 59 chemicals identified as benthic COPCs in the problem formulation was assessed by grouping the COPCs into categories based on the frequency and magnitude of sediment guideline exceedance. This analysis identified 22 COPCs (Groups 1 and 4) that warranted detailed analysis. Based on an initial comparison to

SQS and CSL, total PCBs and BEHP were the two chemicals with the highest exposure potential for benthic invertebrates. CSL exceedances for the other 12 Group 1 COPCs were typically co-located with CSL exceedances of either BEHP or total PCBs. All 59 COPCs are evaluated in the risk characterization (Section A.7.1.1). Using a GIS analysis of multiple chemicals, the following areas with multiple standard or guideline exceedances were identified: south of Harbor Island (RM 0.1), RM 0.3 to 0.6 (east side), Slip 3 and the west side opposite Slip 3 (RM 1.9 to 2.2), most of the east side between Slips 4 and 6, and upstream of Turning Basin 3 (RM 4.8 to 5.5).

As previously discussed, risks to benthic invertebrates from TBT associated with LDW sediments were evaluated in this Phase 1 ERA using a tissue residue approach. Thus, in the exposure assessment, available TBT tissue data were reported and estimated for the LDW based on synoptic sediment and tissue data available from four amphipod tissue samples collected near Kellogg Island. In addition, the limited dataset for crab tissue concentrations of COPCs was used to assess exposure to higher trophic-level benthic invertebrates (Table A-3-19).

Table A-3-19. LDW crab tissue concentrations to use in risk characterization

Chemical	Hepatopancreas (mg/kg ww)	Whole Body [a] (mg/kg ww)
Arsenic	6.98	11.7
Cadmium	0.122	0.037
Chromium	0.083	0.148
Copper	42.9	19.9
Mercury	0.672	0.195
Zinc	19.1	36.1
PCBs	1.65	0.398
TBT	0.0592	0.078

[a] Estimated value based on weighted concentration calculated as 85% of maximum edible meat concentration plus 15% of hepatopancreas concentration.

A.3.3.2 Effects assessment

AETs, which form the basis for SMS standards and DMMP guidelines, were discussed in the effects assessment to provide an indication of the type of effects covered by the sediment standards and guidelines. In addition, results of several studies of sediment toxicity and benthic community characteristics that have been conducted within the LDW in the last 10 years were reviewed. These studies focused on small areas in the LDW. All but two of the sediment toxicity studies were conducted for dredged material characterization, making them unsuitable for an assessment of surface sediment toxicity. Only one of ten samples from the two surface sediment toxicity studies was considered toxic. Although only a few samples have been analyzed for benthic community characteristics, all samples showed some evidence of benthic community alterations relative to reference conditions. The types of benthic

community alterations are characteristic of organic enrichment, and are not necessarily indicative of chemical contamination (Pearson and Rosenberg 1978).

Tissue effects data were also presented for crabs (Table A-3-20), and a threshold screening value for TBT of 3 mg/kg dw was selected for use in the risk characterization to assess risks to benthic invertebrates from TBT exposure.

Table A-3-20. Crab tissue effect concentrations for use in risk characterization

Chemical	Hepatopancreas (mg/kg ww)		Whole Body (mg/kg ww)	
	NOEC	LOEC	NOEC	LOEC
Arsenic	na	na	1.15	na
Cadmium	na	na	4.9[a]	9.5[a]
Chromium	na	na	1	3.2
Copper	na	na	34[b]	na
Mercury	0.99	1.0	na	na
Zinc	42.5	85.6	na	na
PCBs	na	na	23	na
TBT	na	na	0.12	na

na – Effect concentration not available

[a] Based on cadmium in muscle tissue

[b] Based on copper in claw tissue

A.4 Exposure and Effects Assessment: Fish

Three ROCs were selected in the problem formulation to represent the diverse assemblage of fish that utilize the LDW (Section A.2.3.2):

- Wild juvenile chinook salmon (*Oncorhynchus tshawytscha*)
- Bull trout (*Salvelinus confluentus*)
- English sole (*Parophrys vetulus*)

ROC/COPC pairs previously identified in the problem formulation are summarized in Table A-4-1. COPCs were identified for these ROCs based on a comparison of maximum tissue concentrations or maximum estimated metal concentrations in amphipod prey to the lowest NOEC and LOEC data available in the literature. PAHs and PCBs were retained as COPCs for juvenile chinook salmon and English sole because studies from the Pacific Northwest, including the LDW, have shown elevated exposure to PAHs and PCBs (McCain et al. 1990; Varanasi et al. 1993; Stein et al. 1995; Collier et al. 1998; Johnson et al. 1997) and studies have been conducted of potential effects to these fish from PAH and PCB exposure (Arkoosh et al. 1991; Varanasi et al. 1993; Myers et al. 1998a; Arkoosh et al. 1998a,b; O'Neill et al. 1998; Powell et al. in press; Palm et al. in prep). These studies examined survival, growth, reproduction, immunocompetence, lesion prevalence, and biochemical alterations in fish.

Table A-4-1. ROC/COPC pairs to be evaluated for fish

Fish Species	PCBs	PAHs	TBT	DDT	As	Cu	Hg
Juvenile chinook	X	X	X	X	X	X	X
English sole	X	X	X	X	X	X	X
Bull trout	X		X	X	X	X	X

In this ERA, risks to fish were evaluated using two key approaches, depending on the COPC. Exposure to organic compounds and organometallic compounds was assessed through the critical tissue residue approach (i.e., concentrations of COPCs in fish tissue). Exposure to PAHs and metals was evaluated based on dietary dose approximations. The rationale for selecting these approaches was discussed previously in Section A.2.4.6.

Details of the application of these approaches are presented in this section, which is divided into an exposure (Section A.4.1) and effects assessment (Section A.4.2). In addition, relevant field studies conducted in the Puget Sound region are summarized in Section A.4.3. This final section provides a discussion of relevant regional studies involving juvenile chinook salmon and English sole. Data presented in these sections are synthesized in the risk characterization (Section A.7.2) to assess risks to fish in the LDW, and uncertainties are discussed.

A.4.1 EXPOSURE ASSESSMENT

In this section, relevant data were analyzed to determine representative exposure concentrations in the LDW for each ROC/COPC pair identified in Table A-4-1. The primary exposure pathway from sediment-associated chemicals to fish in the LDW is assumed to be through dietary uptake.[67] As described in Section A.2.4, the LDW dataset used to estimate exposure of sediment-associated COPCs to fish consists of a large number of sediment samples, a limited number of tissue, stomach content, and porewater samples, and limited information regarding biomarker measurements in field-collected fish. This section is divided into two subsections to assess exposure based on the approaches discussed above. The first part of this section presents PCB, DDT, TBT, and mercury concentrations in tissue data used for the critical tissue residue approach. The second part of this section presents estimated and measured concentrations in benthic invertebrate prey and measured concentrations of PAHs in stomach contents for dietary approaches, as well as biomarker data as a quantitative indicator of PAH exposure.

A.4.1.1 Tissue data

When available, tissue residues of PCBs, TBT, DDTs, and mercury measured in fish ROCs were used to estimate exposure to these COPCs. This estimate integrates exposure from all pathways to a fish ROC over its home range.[68] Whole-body tissue residue data collected from the LDW were available for juvenile salmon and English sole but not bull trout. The only other fish for which whole body tissue data were available from the LDW is shiner surfperch (Tables A-2-7 and A-4-2).

Twenty individual whole-body juvenile chinook salmon were analyzed for PCBs and DDTs as well as nine composites (NMFS 2002). [69] To maximize the amount of usable data, concentrations in individual fish were combined in order to statistically construct additional composite samples for fish collected near Kellogg Island or Slip 4. In this way, all available data (including individual samples) could be used to estimate exposure of juvenile chinook salmon and also estimate exposure to ROCs that consume juvenile chinook salmon in the LDW. Seven individual whole body concentrations were averaged to construct an additional single composite sample for Slip 4. The 13 individual whole body concentrations from Kellogg Island were allocated to two groups (one of size six and one of size seven) to mathematically construct two additional composite samples for Kellogg Island. Hatchery or wild origin of fish was not considered in allocating fish to composite samples because

---

[67] Direct sediment contact and sediment ingestion were also considered complete significance unknown exposure pathways for English sole (see Figure A-2-2). Sediment ingestion by English sole is addressed in Section A.4.1.2; direct sediment contact will be discussed in the uncertainty assessment (Section A.7.2.2).

[68] Potential uncertainties inherent in the use of whole body burdens to estimate exposure from sediment-associated COPCs in the LDW are discussed in the uncertainty assessment (Section A.7.2.2).

[69] Varanasi et al. (1993) presented whole-body data for composite samples only.

ROCs that consume juvenile chinook salmon would not distinguish between these two types of salmonids in the field.[70]

Table A-4-2. Summary of availability of data regarding PCBs, TBT, DDTs, copper, mercury, and arsenic in fish tissue

ROC	PCBs	TBT	DDTs	COPPER AND ARSENIC [a]	MERCURY
Juvenile chinook	n = 43 (wb, NOAA) [b]	na	n = 43 (wb, NOAA) [b]	na	na
Bull trout	na	na	na	na	na
English sole	n = 3 (wb, KC); n = 30 (fillet, EVS, KC, PSAMP, WSOU)[b]	n = 3 (wb, KC); n = 24 (fillet, EVS, KC, WSOU)	n = 9 (fillet, KC, PSAMP) [b]	n = 3 (wb, KC); n = 9 (fillet, KC, PSAMP) [b]	n = 3 (wb, KC); n = 27 (fillet, EVS, KC, PSAMP) [b]
Perch	n = 3 (wb, KC); n = 12 (fillet, WSOU)	n = 3 (wb, KC); n = 12 (fillet, WSOU)	n = 3 (wb, KC)	n = 3 (wb, KC)	n = 3 (wb, KC); n = 24 (fillet, WSOU)
Rockfish	n = 1 (fillet, PSAMP)	na	n = 1 (fillet, PSAMP)	na	na
Adult salmon (chinook and coho)	n = 138 (fillet, PSAMP)	na	n = 138 (fillet, PSAMP)	n= 34 (fillet, PSAMP)	n= 35 (fillet, PSAMP)

[a] For use in the dietary assessment for piscivorous fish

[b] Twenty-three composite samples (14 from Varanasi et al. [1993] and 9 from NMFS [2002]) and twenty individual samples (from NMFS [2002]) are available. Liver samples are also available.

na – Not available

wb – Whole-body

EVS – Battelle Marine Research Laboratory (1996), EVS (1995), Frontier Geosciences (1996)

KC – King County Water Quality Assessment (1999)

NOAA –Varanasi et al. (1993) and NMFS (2002)

PSAMP – Puget Sound Ambient Monitoring Program (West et al. 2001)

WSOU –Environmental Solutions Group (1999)

Composite sample data were used to assess risk to juvenile chinook salmon consumers, such as bull trout and river otter, because these animals consume a variety of individual juvenile chinook salmon simulating exposure to a composite sample (i.e., a composite more realistically represents the range of chinook these consumers/ predators are eating rather than using individual fish data) . Risk to juvenile chinook salmon was assessed for PCBs and DDTs based on concentrations in individual fish as well because effects on individual fish are of concern due to their ESA status.

[70] To achieve the most conservative exposure point concentrations, the samples with the highest concentrations were allocated to one composite (of size six), although random assignment produced the same result.

Fillet tissue residue data were also available for English sole, rockfish, and adult salmon. As previously discussed, adult salmon tissue data were not used in the fish assessment because adults receive the majority if their chemical exposure, and therefore, tissue residue outside of the LDW; for example, it has been estimated that adult salmon acquire less than 1% of their PCB body burden as juveniles (O'Neill et al. 1998). Rockfish data were not used in the exposure and effects assessment because only one fillet sample was available. Liver samples were also available for juvenile chinook salmon and English sole. These samples were not assessed for risk because appropriate toxicity data for liver tissue concentrations were unavailable.

When whole-body tissue residue data for a particular COPC were not available for an ROC (see Table A-4-2), a series of assumptions were used to estimate body burdens:

- Perch data were used to represent mercury and TBT tissue concentrations in juvenile chinook salmon. These fish have similar habitat preferences and similar diets (see Table A-2-4 of the problem formulation). Additionally, because of their relatively shorter residence time in the LDW, juvenile chinook salmon are not likely to have higher exposure than shiner surfperch.
- No tissue residue data were available for bull trout. Modeled whole-body bull trout tissue concentrations were estimated based on the lower of the maximum or 95% UCL concentration of the whole-body prey data. All fish were considered potential prey; the higher of the perch, juvenile chinook salmon, or English sole tissue residue concentrations was selected for each COPC. Because bull trout are piscivores, predator-prey factors (PPFs) were considered relevant to estimate tissue residues. For mercury, a PPF of 5 was selected based on EPA (1997c); for PCBs, a PPF of 3.5 was selected based on Metcalfe and Metcalfe (1997). A PPF of 3.5 was also selected for DDTs, based on the similarity of its chemical structure to PCBs and on best professional judgment. Use of PPFs is discussed further in the uncertainty assessment (Section A.7.2.2). For TBT, it was assumed that bull trout body burdens were represented by the lower of the maximum or 95% UCL TBT tissue concentration measured in shiner surfperch or English sole from the LDW. This assumption is based on the absence of biomagnification of TBT in aquatic food webs. The higher of the perch or English sole concentration was selected.
- English sole fillet data were used to estimate DDTs (DDT, DDE and DDD) exposure for English sole. Whole body DDT tissue residues are likely to be somewhat higher than fillet tissue residues. Uncertainties inherent in the use of fillet data are discussed in the uncertainty assessment (Section A.7.2.2.2).

A summary of available whole-body tissue residue data and estimated tissue residue concentrations using the methods described above is presented in Table A-4-3. The 95% UCL on the mean of the data and its range are presented; the lower of the 95% UCL or maximum concentration will be used to represent exposure to a particular ROC in the risk characterization (EPA 1992).

Table A-4-3. Measured and estimated chemical concentrations in LDW whole-body fish tissue (µg/g ww)

COPC	Species	Sample 1[a]	Sample 2[a]	Sample 3[a]	95% UCL[b]	No. of Samples	No. of Fish per Composite
Mercury	Perch	0.071	0.075	0.088	0.093	3	10
	English sole[c]	0.064	0.076	0.0602	0.0803	3	20
	Modeled bull trout[d]	0.44					
TBT (as ion)	Perch	0.12	0.16	0.179	0.206	3	10
	English sole[c]	0.0118	0.019	0.0122	0.0212	3	20
	Modeled bull trout[e]	0.179					
Total PCBs[f]	Perch	0.35	0.519	0.616	0.721	3	10
	English sole[c]	0.721	2.306	1.43	2.825	3	20
			Min	Max	95% UCL	N[g]	
Total PCBs	Juvenile chinook [h,i] (composite)		0.17	0.260	0.160	26[m]	2–10
	Juvenile chinook[h] (individual)		0.14	0.750	0.266	20	1
	Modeled bull trout[j]	8.07					
DDTs[k] (total calculated)	Juvenile chinook[h] (composite)		0.003	0.049	0.031	26[m]	2–10
	Juvenile chinook[h] (individual)		0.002	0.021	0.018	20	1
	English sole fillet		0.001	0.011	0.0069	9	5–20
	Modeled bull trout[l]	0.11					

[a] Samples collected for King County Water Quality Assessment (1999)

[b] 95% UCL on the mean concentration

[c] Whole body concentrations estimated from carcass and viscera data (see Section A.7.2.2.2)

[d] Equivalent to maximum measured perch tissue multiplied by a PPF of 5

[e] Equivalent to maximum measured perch tissue

[f] Sum of detected Aroclors

[g] Number of samples

[h] Sum of PCB homologues (trichlorobiphenyls through decachlorobiphenyls)

[i] Data from Varanasi et al. (1993) and NMFS (2002)

[j] Equivalent to maximum English sole tissue concentration multiplied by a PPF of 3.5

[k] Sum of detected concentrations of DDT, DDD, and DDE

[l] 95% UCL concentration in juvenile chinook salmon multiplied by a PPF of 3.5

[m] Chemical data were available for 23 composite samples (14 from Varanasi et al. [1993] and 9 from NMFS [2002]). An additional three composite samples were statistically constructed using samples of individuals, as discussed in Section A.4.1.1.

A.4.1.2 Dietary exposure

This section presents the dietary approach used to estimate exposure of the three fish ROCs to arsenic and copper (Section A.4.1.2.1) and PAHs (Section A.4.1.2.2). PAH exposure is also assessed qualitatively through the use of the biomarkers biliary fluorescent aromatic hydrocarbons (FACs) and hepatic DNA adducts.

A dietary exposure approach rather than a critical tissue residue approach is preferable for COPCs that are highly compartmentalized or metabolized by fish, such as copper, arsenic, and PAHs. Copper, an essential nutrient, is highly compartmentalized by fish (Adams et al. 2000). Arsenic uptake is also metabolically regulated, and does not increase above a certain species-specific threshold (Maher and Butler 1988). PAHs are highly metabolized by fish (Varanasi 1989).

The dietary exposure approach requires an approximation of the COPC concentration in an ROC's prey. Stomach content analysis of juvenile chinook salmon from the LDW indicate that they typically consume epibenthic invertebrates such as amphipods, worms, and clam siphons, as well as flying insects and other drift organisms (Cordell et al. 1997). Bull trout are suspected to prey on juvenile salmonids and juvenile shiner surfperch based on the capture of bull trout in the LDW during times of high juvenile shiner surfperch abundance (Shannon 2001; Section A.2.2.3.2). English sole consume epibenthic invertebrates such as amphipods, small crabs, small sea stars, small brittle stars, marine worms, and mollusks (Hart 1973; Feder 1980). English sole may also incidentally ingest sediment because of the nature of their prey and their close proximity to sediment. The above ROCs' diets were considered, as described in the following sections, to determine appropriate prey for calculating exposure in the LDW.

A.4.1.2.1 Arsenic and copper

Available LDW data for arsenic and copper in potential fish ROC prey species include tissue measurements of amphipods, crabs, and mussels as well as tissue residue in perch. These data are summarized in Table A-4-4.

Of the available prey data for arsenic and copper, the prey of juvenile chinook salmon was best represented by the amphipod data because these fish are not known to consume mussels, crabs, or large fish. Of the available prey data, bull trout were assumed to consume adult shiner surfperch, although observations in the LDW indicate they are more likely to consume juvenile shiner surfperch.[71] Of the available prey data, English sole are known to consume epibenthic invertebrates, such as amphipods, as well as small crabs.

[71] Concentrations in adult perch are expected to be higher than or equal to those in juvenile perch.

Table A-4-4. Available tissue data for fish prey items in the LDW

Prey Item	Arsenic (mg/kg dw)	Copper (mg/kg dw)
Amphipods[a]		
maximum	8.21	167
mean	6.61	104
95% UCL on the mean	8.19	166
n	4	4
Mussels[b]		
maximum	4.46	7.13
mean[c]	3.48	4.87
95% UCL on the mean	3.78	5.34
n	22	22
Dungeness crabs[a,d]		
maximum	60.3	90.6
mean[e]	47.0	89.4
n	2	2
Whole body perch[b]		
maximum	5.79	9.13
mean	5.30	6.02
95% UCL on the mean	6.72	10.6
n	3	3

[a] Dry weight concentrations calculated based on total solids measured in each sample.

[b] Dry weight concentrations calculated based on an assumed 24% solids (based on English sole tissue measurements).

[c] Weighted average concentration; first the average concentration of each station was calculated, and then the average of these numbers was calculated.

[d] Concentrations represent weighted values using 85% edible meat and 15% hepatopancreas (n = 1) concentrations.

[e] For crab tissue, a simple arithmetic mean rather than a 95% UCL on the mean was calculated because only two samples were available.

Although tissue residue data are not available for small crabs, it was assumed that the available Dungeness crab tissue residue data were the most appropriate available data and should result in a conservative estimate of exposure. Whole body tissue residues in Dungeness crab were based on a weighted mean of hepatopancreas and edible meat data calculated assuming 15% hepatopancreas and 85% edible meat by weight, as shown in Equation 4-1.

$$C_{wb} = (XC_{edible\ meat}) \times (XC_{hepatopancreas})$$ *Equation 4-1*

Where:

X	=	proportion of a given tissue
C_{wb}	=	whole body concentration
$C_{edible\ meat}$	=	edible meat concentration
$C_{hepatopancreas}$	=	hepatopancreas concentration

Other prey items for which no representative prey tissue data were available are discussed in the uncertainty assessment (Section A.7.2.2.2).

Bull Trout Exposure to Arsenic and Copper

Shiner surfperch tissue data were used to approximate dietary exposure to bull trout based on the assumed piscivorous dietary preferences discussed above and the available data. The lower of the maximum or 95% UCL on the mean shiner surfperch concentration will be used to estimate bull trout exposure to arsenic and copper in the risk characterization. For both chemicals, the maximum concentration was lower than the 95% UCL on the mean so the maximum concentrations (5.79 mg/kg dw for arsenic and 9.13 mg/kg dw for copper) are used in the risk characterization (Section A.7.2.1) to represent dietary exposure of bull trout to these chemicals in the LDW.

Juvenile Chinook Exposure to Arsenic and Copper

Because amphipods constitute a major component of the juvenile chinook salmon diet, and no other data regarding chemical concentrations in prey were available, amphipod tissue data were used to approximate dietary exposure to juvenile chinook salmon in the LDW. The lower of the maximum or 95% UCL on the mean arsenic and copper concentration in amphipod tissue will be used to estimate exposure in the risk characterization (Section A.7.2.1). These concentrations were 8.19 and 166 mg/kg dw for arsenic and copper, respectively (Table A-4-4).

In addition, because amphipod data were collected from a limited area in the LDW (near Kellogg Island), and the home range of amphipods is very small relative to the spatial heterogeneity of the sediment contamination in the LDW, a BAF calculated from the available tissue and sediment data was used to estimate amphipod tissue COPC concentrations in other LDW locations. It is recognized that use of this BAF introduces considerable uncertainty into the exposure calculation because of the limited dataset, the potential for variable BAFs because of variable COPC concentrations and bioavailability, and the various benthic invertebrate species that may actually be consumed by juvenile chinook (see Section A.7.2.2). Discussed below are the data used to calculate the BAFs for arsenic and copper and how they were applied to estimate exposure concentrations.

Because no data were available to definitively outline areas of foraging preference for juvenile chinook salmon, it was assumed that these fish forage throughout the LDW without preference to particular areas. Information on juvenile chinook salmon in estuarine environments suggests that they predominantly occupy the margins of

water courses and shallow water habitats. However, this information has not been verified for habitat use in the LDW. Thus, SWA copper and arsenic concentrations in surface sediment from the entire LDW, as well as for the LDW excluding the deep-water shipping channel,[72] were calculated for use in estimating concentrations in amphipods, as discussed below. SWA-based estimates of amphipod concentrations are reasonable for use in estimating dietary exposure to juvenile chinook salmon because the salmon spatially average their dietary exposure through their consumption of amphipods as they migrate through the LDW. Due to heterogeneous sediment sampling efforts for different areas of the LDW, SWA concentrations provide a more realistic representation of the distribution of sediment concentrations than statistics that do not take into account the spatial distribution of the sampling effort.

The SWA sediment concentration was determined from the weighted distribution of all measurements, with each weight equal to the area of its associated Thiessen polygon as a fraction of the total area for which samples have been collected. To account for spatial variability in sampling density (i.e., greater coverage near locations where contamination is known or expected to exist), Thiessen polygons are widely used as the nonparametric method of spatial analysis. The Thiessen polygon associates each point in a plane with the closest neighbor for which a measurement is available (Burmaster and Thompson 1997). In effect, this algorithm assumes that the concentration at any point where measurements have not been made is the same as the concentration in the sample closest to that point. The SWA concentration is a weighted average of all measurements, with each weight equal to the area of its associated polygon as a fraction of the total area for which samples have been collected. For a given chemical, the concentration measured at that sample location is assigned to the polygon. In cases where multiple samples were collected at a single location, during one or multiple events, the mean concentration of all samples is assigned to the polygon. Undetected values were assigned a value equal to one-half the detection limit. Polygons were defined by the distance to the nearest sampling locations. The northern and southern extents of the surface sediment sampling events included in this analysis were defined by the southern edge of Harbor Island and the southern boundary of the GIS shape files created by Weston for the EPA Site Inspection (Weston 1999), respectively. Similar boundaries were created along the upland margins of the intertidal sampling locations.

As shown in Table A-4-5, amphipod tissue concentrations estimated using SWA sediment concentrations were similar to those measured near Kellogg Island. This is due to the similarity between SWA sediment concentrations of arsenic and copper and sediment concentrations near Kellogg Island where amphipods were collected. The similarity in these sediment concentrations supports the use of the measured amphipod tissue data rather than the BAF modeled data to estimate dietary exposure.

[72] No difference in SWA concentrations of arsenic or copper was found when the deep-water shipping channel was excluded because relatively few samples were available from the channel.

Table A-4-5. Estimated amphipod tissue concentrations in LDW

Chemical	Avg KI Amphipod (mg/kg-dw)	Avg KI Sediment (mg/kg-dw)	BAF KI	Avg W. Mar. Amphipod (mg/kg-dw)	Avg Sed W. Mar (mg/kg-dw)	BAF W. Mar	LDW Sediments SWA (mg/kg-dw)	SWA Estimated Amphipod Tissue (mg/kg-dw)
Arsenic	5.5	5.7	0.96	7.7	12	0.64	12	7.7 - 12
Copper	61	20	3.0	147	65	2.3	58	133-174

Note: SWA sediment concentrations were also calculated for the LDW excluding the deep-water shipping channel, with no difference in results due to the relatively few data from the channel.

KI – Kellogg Island

BAF – bioaccumulation factor

W Mar – West Marginal Way

SWA – spatially weighted average concentration

Thus, the lower of the maximum or 95% UCL arsenic and copper concentration in measured amphipod tissue was selected for use in the risk characterization (Section A.7.1). These concentrations were 8.2 and 166 mg/kg dw for arsenic and copper, respectively (Table A-4-4). Uncertainties associated with the use of these data are discussed in the uncertainty assessment (Section A.7.2.2).

English Sole Exposure to Arsenic and Copper

Site-specific data were not available to estimate the relative proportions of prey in the English sole diet. In addition, no data were available to estimate exposure through incidental sediment ingestion. Thus, best professional judgment was used to estimate sediment ingestion at 10% of the diet, and to divide the remaining 90% between benthic invertebrates, such as epibenthic amphipods and crabs (i.e., each assumed to account for 45% of the English sole diet), according to the following equation:

$$C_{food} = \Sigma_i X_i C_i = X_{sed}C_{sed} + X_{amp}C_{amp} + X_{crab}C_{crab} \qquad \textit{Equation 4-2}$$

Where:

C_{food} = arsenic or copper concentration in the English sole diet (mg/kg dw)

X_i = portion diet of a particular food item (assumed to be 0.1 for sediment, 0.45 for amphipods, and 0.45 for crabs) (unitless)

C_i = arsenic or copper concentration in the prey item (mg/kg dw)

C_{sed} was represented by the SWA sediment concentration (Table A-4-5) because English sole are likely to have a relatively large home range.[73] C_{amp} was the lower of the maximum or 95% UCL on the mean concentration estimated in amphipods (Table A-4-4). C_{crab} was the maximum concentration of the two samples analyzed for

[73] Effects of this assumption are addressed in the uncertainty assessment (Section A.7.2.2.2).

arsenic or copper in Dungeness crab (assuming 15% hepatopancreas by weight) (Table A-4-4). These assumptions resulted in the following dietary concentrations:

Concentration of As in sole prey = (0.1)(12) + (0.45)(8.19) + (0.45)(60.3)= 32 mg/kg dw

Concentration of Cu in sole prey = (0.1)(58) + (0.45)(166) + (0.45)(90.6) = 121 mg/kg dw

Thus, in the risk characterization dietary exposure to English sole was represented by 32 mg/kg dw arsenic and 121 mg/kg dw copper.

A.4.1.2.2 PAHs

PAHs were selected as COPCs for both juvenile chinook salmon and English sole in the problem formulation. PAHs were not selected as a COPC for bull trout based on minimal exposure because of their piscivorous diet. In this section, dietary exposure to PAHs by juvenile chinook salmon was assessed using stomach content data from juvenile chinook salmon collected in the LDW. Dietary exposure to PAHs by English sole was assessed using an approach similar to that presented above for arsenic and copper.

Juvenile Chinook Salmon Dietary Exposure to PAHs

PAH concentrations in stomach contents of juvenile chinook salmon collected from the LDW have been reported in McCain et al. (1990), Varanasi et al. (1993), and Arkoosh et al. (1998b) (Table A-4-6). All fish reported in these three studies were collected by beach seine near Kellogg Island. Dietary composition was not noted in any of these studies. Stomach content chemical data integrate the recent diet of collected fish, all of which may not be associated with sediments.[74]

The mean total PAH concentration in the stomach content of juvenile chinook salmon from the LDW was 20 mg/kg ww (n = 8), the maximum concentration was 74 mg/kg ww, and the 95% UCL on the mean concentration was 33 mg/kg ww[75] (PAH assemblages measured in each of studies are footnoted in Table A-4-6). PAH stomach content concentrations were highly variable and ranged over an order of magnitude within a single year. The data variability may result from differences in fish collection locations, exposure duration, diet, and possibly exposure to concentrated substrates like creosote. Variability in the 1990 stomach content data from Commencement Bay was of similar magnitude with PAH concentrations ranging from 3.6 to 106 mg/kg ww (Varanasi et al. 1993).

Because the 95% UCL on the mean total PAH concentration of 33 mg/kg ww[76] is lower than the maximum concentration, it was used in the risk characterization

---

[74] Uncertainties in the use of stomach content data to estimate exposure to juvenile chinook salmon from sediment-associated COPCs in the LDW are discussed in the uncertainty assessment (A.7.2.2.2).

[75] Data from Arkoosh et al. (1998) were not included in this calculation because the underlying data were not available (only mean and standard deviation concentrations were reported). These data will be included in the final Phase 1 ERA if they become available.

[76] Equivalent to 165 mg/kg dw based on an assumed 20% solids.

(Section A.7.2) to represent dietary PAH exposure to juvenile chinook salmon in the LDW.

Table A-4-6. PAH concentrations in juvenile chinook salmon stomach contents (mg/kg ww)[a,b]

Study	Site	Collection Date	LPAHs	HPAHs	LPAH + HPAH
McCain et al. 1990 [c]	LDW	June 12, 1986 rep1	13.9	7.8	21.7
McCain et al. 1990 [c]	LDW	June 12, 1986 rep2	7.0	3.9	10.7
Varanasi et al. 1993 [d]	LDW	May 23, 1989	0.75	46	47
Varanasi et al. 1993 [d]	LDW	June 1, 1989	14	5.0	19
Varanasi et al. 1993 [d]	LDW	June 7, 1989	1.1	3.5	4.6
Varanasi et al. 1993 [d]	LDW	May 24, 1990	0.70	4.6	5.3
Varanasi et al. 1993 [d]	LDW	June 1, 1990	1.4	2.8	4.3
Varanasi et al. 1993 [d]	LDW	June 18, 1990	41	33	73
Arkoosh et al. 1998b	LDW	June 11, 14, 1993	2.9 (3.0)[e]	6.2 (3.8)	9.1
Arkoosh et al. 1998b	LDW	June 1, 1994	2.1 (0.9)	2.8 (1.7)	4.9

[a] Only concentrations of Washington SMS-defined PAHs were reported (SMS Chapter 173-204 WAC). LPAHs (low-molecular-weight PAHs) = naphthalene, 2-methylnaphthalene, acenaphthylene, acenaphthene, fluorene, phenanthrene, anthracene; HPAHs (high-molecular-weight PAHs) = fluoranthene, pyrene, benz(a)anthracene, chrysene, benzo(b)fluoranthene, benzo(j)fluoranthene, benzo(k)fluoranthene, benzo(a)pyrene, indeno(1,2,3-c,d)pyrene, dibenz(a,h)anthracene, benzo(g,h,i)perylene.

[b] Only detected concentrations were included in summed PAH concentrations.

[c] Concentrations were reported as "estimated dry weight" based on an estimated 20% solids. Wet weight concentrations were recalculated from this ratio. LPAHs reported did not include acenaphthylene. HPAHs reported did not include benzo(b)fluoranthene, benzo(k)fluoranthene, indeno(1,2,3-cd)pyrene, dibenz(a,h)anthracene, benzo(ghi)perylene.

[d] LPAHs reported did not include 2-methylnapththene or anthracene.

[e] Numbers in parentheses represent standard deviations

English Sole Dietary Exposure to PAHs

Stomach content PAH concentration data were not available for English sole in the LDW. Therefore, an approach for estimating PAH dietary exposure analogous to that used to estimate arsenic and copper exposure was used (see Section A.4.1.2.1). In Section A.4.1.2.1, best professional judgment was used to estimate sediment ingestion at 10% of the diet, and the remaining 90% of the diet was divided between epibenthic amphipods and crabs (each was assumed to constitute 45% of the English sole diet). However, because PAHs were not detected in the available crab tissue data, the sole diet in this section was assumed to consist of 10% sediment and 90% amphipods in order to avoid the uncertainties associated with the use of detection limit concentrations for PAHs. Therefore, total PAH concentrations in English sole diet were estimated using the following equation:

$$C_{food} = X_{sed}C_{sed} + X_{amp}C_{amp} \qquad \textit{Equation 4-3}$$

Where:

C_{food} = PAH concentration in English sole diet (mg/kg dw)

X_{food} and X_{amp} = the fraction of the diet represented by sediment (0.1) and amphipods (0.9)

C_{sed} = represented by the SWA sediment concentration of total PAHs (4.4 mg/kg dw) because English sole are likely to have a relatively large home range

C_{amp} = C_{sed} normalized for organic carbon and multiplied by the BSAF calculated in Table A-4-7

The only amphipod PAH data available to estimate dietary exposure were from two locations in the vicinity of Kellogg Island (West Marginal Way) (discussed in Section A.4.1.2.1). A biota-sediment accumulation factor (BSAF) was calculated from the available amphipod data and co-located sediment data using the following equation:

$$BSAF = \frac{\text{Biota (mg/kg lipid)}}{\text{Sediment (mg/kg OC)}} \qquad \textit{Equation 4-4}$$

Estimated tissue concentrations are presented in Table A-4-7. These BSAFs are consistent with data reported in Thomann and Komlos (1999) for PAH uptake by crayfish.

Table A-4-7. Amphipod TPAH BSAF calculated using synoptic sediment and amphipod data from near West Marginal Way

PAH Sum	Amphipod TPAH Concentration (mg/kg lipid)	Sediment TPAH Concentration [a] (mg/kg OC)	BSAF
Sum of PAHs detected in tissue	4.7[b]	160[c]	0.029
TPAH	10[d]	440[e]	0.023

[a] Average of three replicate sediment samples collected near West Marginal Way

[b] Only one of two samples had detectable PAHs, and only pyrene and fluoranthene were detected in that sample

[c] Sum of pyrene and fluoranthene concentrations in sediment, OC-normalized

[d] Sum of detected PAH concentrations and ½ detection limits for undetected PAHs (total of 16 PAH compounds)

[e] Sum of detected PAH concentrations; the few PAH compounds that were undetected were not included in the sum because the detection limits were not available

Using a spatially weighted and organic carbon (OC)-normalized sediment TPAH concentration (400 mg/kg OC), a lipid content of 5% (measured in amphipods collected near West Marginal Way), and a moisture content of 80%, C_{amp} was calculated using the higher of the two BSAFs.

$$C_{amp} = BSAF \times C_{sed} \times 0.05 = 0.60 \text{ mg/kg ww} = 3.0 \text{ mg/kg dw}$$

Using the equation above, C_{food} is 3.1 mg/kg dw. This concentration is used in the risk characterization to represent dietary exposure of English sole to total PAHs.

PAH Biomarkers of Exposure

Another method by which PAH exposure in the LDW can be assessed is through the use of biomarkers. Biliary FACs and hepatic DNA adducts are biomarkers useful as an indicator of PAH exposure. Biliary FACs provide a semi-quantitative measure of PAH metabolites that are in the process of being excreted from the organism (Varanasi et al. 1989). This biomarker is specific to PAH exposure and metabolism. DNA adducts result from the covalent bonding of a contaminant (e.g., PAHs, aflatoxins, aromatic amines) to DNA. However, adduct formation may not always be correlated with contaminant exposure because of other factors such as DNA repair and natural causes of adduct formation (Shugart et al. 1992). DNA adducts are reported to be a longer-term indicator of exposure than biliary FACs (Varanasi et al. 1993).

Interpretation of biomarker levels in field-exposed fish can be complicated by a number of variables. For example, biliary FACs in English sole have been shown to be strongly affected by feeding status (Collier and Varanasi 1991). Measured on a per mL bile basis, biliary FAC levels decreased significantly in exposed and control animals that were feeding and increased in animals not feeding. However, normalization to bile protein is believed to compensate for feeding status to some extent (Collier and Varanasi 1991). Other factors, such as exposure to other co-occurring contaminants (e.g., co-planar PCBs) can also impact results (Varanasi et al. 1989). Thus, biliary FACs and DNA adducts are interpreted to provide a qualitative indication of elevated PAH exposure to fish within the LDW relative to reference areas.

Juvenile Chinook Salmon

Biliary FACs and DNA adducts have been measured in juvenile chinook salmon collected from the LDW, in hatchery fish prior to their release from the Green River Hatchery to the LDW, and in the Nisqually estuary, a regional reference site (Table A-4-8).

Data from Arkoosh et al. (1998b), McCain et al. (1990), and Varanasi et al. (1993) showed that biliary FACs from LDW juvenile chinook salmon were higher than biliary FACs from fish collected from the relatively uncontaminated Nisqually estuary. Data from Varanasi et al. (1993) show that DNA adducts in LDW juvenile chinook salmon were significantly higher than pooled DNA adduct data representing fish collected from three reference sites: the Nisqually estuary, the Green River hatchery, and the Kalama Creek hatchery (in the Nisqually River system).

Consistent with the variability seen in PAH concentrations in juvenile chinook salmon stomach contents (discussed in this section under dietary exposure), data from Arkoosh et al. (1998b) showed that levels of biliary FACs in LDW chinook, even from a single sampling event, had a high degree of variability (Table A-4-8). As discussed above under dietary exposure, the apparent variability in PAH exposure may be the result of differences in fish collection locations, exposure duration, diet, and possibly exposure to concentrated substrates like creosote.

Table A-4-8. Biliary FACs and DNA adducts detected in juvenile chinook salmon from the LDW and reference areas

Citation	Location	Date Collected	Treatment	DNA Adducts (nmol adducts/ mol bases)	Biliary FACs (ng BAP eq/mg bile protein)
Arkoosh et al. 1998b	LDW	1993 1994	field	not analyzed	2,400 (1,100) [a, b] 4,300 (1,100) [a]
Arkoosh et al. 1998b	GR hatchery	1993 1994	hatchery	not analyzed	1,110 (23) 2,400 (310) [c]
Arkoosh et al. 1998b	Nisqually estuary	1993 1994	field	not analyzed	230 (64) 550 (110)
McCain et al. 1990	LDW	June 1986	field	not analyzed	0.89 – 1.7 µg BaP eq/g bile ww [d]
McCain et al. 1990	Nisqually estuary	1986	field	not analyzed	0.04-0.07 µg BaP eq/g bile ww [d]
McCain et al. 1990	Nisqually hatchery	1986	hatchery	not analyzed	0.06-0.23 µg BaP eq/g bile ww [d]
Varanasi et al. 1993	LDW	June 1989 June 1990	field	12 [a, e] 6 [a, e]	450 [e] 550 [e]
Varanasi et al. 1993	GR and NR hatcheries	1989 1990	hatchery	7 [e] 5 [e]	150 [e] 150 [e]
Varanasi et al. 1993	Nisqually estuary	1989 1990	field	4 [e] 4 [e]	80 [e] 80 [e]

GR – Green River

NR – Nisqually River

[a] Significantly different from Green River hatchery and Nisqually estuary

[b] Number in parentheses is standard deviation

[c] Significantly different from Nisqually estuary and hatchery

[d] Biliary FACs were not normalized to bile protein

[e] Concentration approximated from graph

The data from Arkoosh et al. (1998b) also showed that both the 1993 and 1994 Green River hatchery fish had elevated biliary FACs relative to fish from the Nisqually estuary. Thus, some exposure of juvenile chinook salmon to PAHs may occur at the hatchery prior to release of fish to the Green River.

In summary, levels of biliary FACs and DNA adducts from juvenile chinook salmon collected in the LDW relative to those collected from reference sites provide a qualitative indication that PAH exposure in the LDW was higher than would be expected in an uncontaminated site within the region. Biliary FAC data also suggest that some portion of this exposure may have occurred in the hatchery. Because levels of biliary FACs and DNA adducts cannot be quantitatively linked to a specific PAH exposure concentration, these data are discussed in a qualitative manner in the risk characterization (Section A.7.2.1).

English Sole

Biliary FACs and DNA adducts have been measured in English sole collected in the LDW. Table A-4-9 presents the available data and provides a comparison to biliary FAC and DNA adduct levels in fish from reference areas.

Table A-4-9. Biliary FACs and DNA adducts detected in field-collected English sole from the LDW and reference areas

Citation	Location	Date Collected	DNA adducts (nmol adducts/mol bases)	Biliary FACs (ng BAP eq/mg bile protein)
Casillas et al. 1991	LDW	December – January 1987 and 1988	na	240 ± 130
	Reference: Port Susan	December – January 1987 and 1988	na	200 ± 100
Stein et al. 1992	LDW	May 1988	140 ± 27 (n= 4)[a]	250 ± 52 (n=4)
	Reference: Polnell Point	May 1988	35 ± 0.74 (n= 3)	340 ± 110 (n=4)
Collier et al. 1992	LDW	October and November 1986	na	690 ± 160 (n = 12)[a]
	Reference: Saratoga Passage	October and November 1986	na	70 ± 11 (n=12)
Myers et al. 1998b	LDW	November 1987	na	310 (n=4)
	Reference: Polnell Point	November 1987	na	12 (n=4)
	LDW	January 1988	na	380 (n=4)
	Reference: Polnell Point	January 1988	na	240 (n=4)
	LDW	August 1988	na	360 (n=4)
	Reference: Polnell Point	August 1988	na	180 (n=4)
	LDW	October 1988	na	590 (n=4)
	Reference: Polnell Point	October 1988	na	99 (n=4)

na – Not available

[a] Significantly different from reference

Casillas et al. (1991) measured biliary FAC concentrations in vitellogenic female English sole collected from the LDW and a reference site (Port Susan). No significant difference was reported between the biliary FAC concentrations measured in samples from the two locations.

Similarly, Stein et al. (1992) found no significant difference between the biliary FAC concentrations in fish collected from the LDW and those in reference fish from Polnell Point. However, significantly higher concentrations of DNA adducts were measured in fish from the LDW relative to reference fish. Collier et al. (1992) reported significantly higher biliary FAC concentrations in LDW fish relative to those collected from Saratoga Passage, a reference site. Consistently higher biliary FAC levels were also measured in fish collected from the LDW compared to reference fish collected from Polnell Point, although statistical analyses were not performed (Myers et al. 1998b).

Johnson et al. (1997) measured biliary FAC concentrations in English sole collected from the LDW, Eagle Harbor, and two reference locations (Port Susan and Sinclair Inlet). Biliary FAC concentrations measured in fish collected from the LDW were

reported to be significantly higher than concentrations for the reference fish. The results of this study are not reported in Table A-4-10 because biliary FAC data were presented in a log-scale figure and it was not possible to estimate biliary FAC concentrations accurately from the figure.

In summary, biliary FAC concentrations were significantly higher in English sole collected from the LDW than from reference areas, Polnell Point and Saratoga Passage, in three of the five studies. These results suggest that PAH exposure to English sole was higher in the LDW than in reference areas, although considerable variability exists. The results from Myers et al. (1998b) also suggest that biliary FAC concentrations were highly variable seasonally. The results of a single study of DNA adducts in English sole from the LDW also suggested higher exposure in the LDW than in a reference area. The available DNA adduct and biliary FAC data serve as only qualitative indicators of English sole exposure to PAHs in the LDW, and will be used qualitatively in the risk characterization.

A.4.2 Effects Assessment

In this section, laboratory studies reported in the toxicological literature were reviewed for COPCs identified in the problem formulation. Toxicological studies reporting fish tissue residues of PCBs, TBT, DDTs, and mercury associated with potential effects were reviewed, in addition to dietary studies involving arsenic, copper, and PAHs. Effects data presented in this section are assessed in combination with exposure data (Section A.4.1) in the risk characterization (Section A.7.3). The results of several site-specific and region-specific studies that have addressed toxicological effects on juvenile chinook salmon and English sole are presented in Section A.4.3. These studies were addressed separately because they involve exposure to contaminant mixtures and subsequent assessment of effect, thus no chemical-specific NOECs or LOECs can be determined from these studies.

To develop toxicity reference values (TRVs), the toxicity literature was searched and single chemical toxicity data (or single chemical mixture data in the case of PAHs and PCBs) for fish were compiled, including no or lowest observed effects concentrations (NOECs or LOECs). The selection of studies relating fish tissue residues to effects was based on a search of electronic databases including Environmental Residue Effects Database (ERED; ACOE 2002), NOAA (Beckvar et al. 1996), and a compilation of tissue residue LOECs and NOECs (Jarvinen and Ankley 1999), and the primary literature using the electronic BIOSIS database for papers published from 1998 to the present. The selection of studies relating dietary concentrations of COPCs to effects was based on the electronic database AQUIRE, and a search of the primary literature using the electronic BIOSIS database from 1998 to the present.

Guidelines for the selection of NOECs and LOECs were:

- Taxonomic similarity to ROCs; i.e., fish of the same family as juvenile chinook salmon and bull trout (for nonbiomagnifying chemicals) were given preference,

after which all fish were given equal preference. For English sole taxonomic similarity was not a criterion because this ROC represents fish of several different families. For bull trout, taxonomic similarity was not a criterion for evaluation of chemicals that biomagnify (i.e., mercury, DDTs, and PCBs), because for these COPCs, bull trout was selected to represent fish of families that may be more sensitive to these chemicals.

- Relevance of exposure conditions within the study relative to those in the LDW (including dose and route of exposure)
- Studies that incorporated intraperitoneal (IP) injection as the exposure route were not used for selection of NOECs or LOECs because the injected chemical dose cannot be related to environmental exposure of the fish. IP injection is commonly used in investigations to assess potential effect mechanisms. However, the dose response relationship found in IP injection studies is frequently different from studies conducted using more environmentally relevant exposure mechanisms. For example, Varanasi (1989) found that production of PAH metabolites in fish livers was greater in fish exposed through IP injection than through dietary exposure. These data suggest that the PAH dose received through IP exposure was transported more rapidly to the liver than the dietary dose, resulting in faster accumulation in the liver relative to the rate of metabolism of the compound. Thus, IP injection studies were not used to determine NOECs and LOECs, and were not discussed or presented in this section unless no other data were available for a given COPC and endpoint.
- Assessment endpoints identified for juvenile chinook salmon were survival and growth. Survival following immunological challenge was also evaluated for PCBs and PAHs because it has been suggested that juvenile chinook salmon PCB and PAH exposure in the LDW results in increased mortality due to reduced immunocompetence (Varanasi et al. 1993; Arkoosh et al. 1998b). Juvenile chinook salmon outmigrate through the LDW and are present as fry/smolts. Spawning and early life stages of these fish do not occur in the LDW.[77] Furthermore, adult salmon are believed to acquire over 99% of their body burden of chemicals outside of the LDW (O'Neill et al. 1998), and thus most of the potential maternal transfer of chemicals would not result from LDW exposure. Therefore, toxicological studies investigating reproduction or those involving eggs or embryos were not evaluated for this ROC.
- Assessment endpoints identified for bull trout were survival, growth, and reproduction. Bull trout are occasionally present in the LDW as adult or subadult fish; their remaining habitat is unknown. Spawning and early life stages of bull trout do not occur in the LDW. However, chemical exposure within the LDW may affect reproduction and development in eggs spawned

[77] On rare occasions, alevins are washed into the LDW during periods of high flow (see Section A.4.3 in the problem formulation).

elsewhere. Thus, for arsenic, copper, and TBT, only studies in which adult fish were exposed to COPCs were considered for selection of reproduction TRVs for bull trout. Use of the LDW by other piscivores represented by this ROC could include spawning and early life stages. Therefore, for mercury, DDT and PCBs, all reproduction studies (including exposure to eggs and embryos) were considered for selection of TRVs because, for these biomagnifying chemicals, the bull trout ROC represents piscivorous fish that may reproduce in the LDW.[78]

- Assessment endpoints identified for English sole were survival, growth, and reproduction. For PAHs, endpoints such as cancerous lesions were evaluated because PAHs have been linked to increased lesion prevalence in English sole from the LDW (Malins et al. 1984; Rhodes et al. 1987; Johnson and Landahl 1994). Research suggests, however, that increased lesion prevalence does not contribute to mortality of individuals or population effects (Johnson and Landahl 1994). All life stages of English sole are present in the LDW except for their seasonal migrations to Puget Sound for spawning. Because English sole (and fish represented by this ROC) may spend a significant portion of their lifetime in the LDW, maternal transfer may contribute to chemical burdens in eggs. Therefore, exposure within the LDW may affect reproduction and development in eggs spawned in the LDW or elsewhere. Thus, toxicological studies involving reproduction and eggs or embryos were evaluated for this ROC.

The remainder of this section presents a COPC-specific review of the toxicological literature for COPCs identified in the problem formulation. Laboratory studies were examined for ranges of NOECs and LOECs, and specific NOECs and LOECs were recommended for use in the risk characterization (Section A.7.3). COPC/endpoint pairs for which no toxicity data were available are addressed in the uncertainty assessment (Section A.7.2.2).

A.4.2.1 Arsenic

Arsenic, a COPC for all three fish ROCs, was evaluated in this ERA from a dietary perspective (Section A.4.1.2.1). Thus, the literature was searched for laboratory studies involving dietary exposures of arsenic associated with survival, growth, and reproductive endpoints. Six relevant studies were identified, some of which presented results of several separate experiments (Table A-4-10). In total, 10 LOECs and 7 NOECs were identified. For each ROC, these studies were reviewed to identify a suitable NOEC and LOEC for use in the risk characterization (Section A.7.2.1). The lowest relevant LOEC and the highest relevant NOEC below it were selected for each ROC and endpoint.

[78] It is likely that the Phase 2 ERA piscivorous fish will be represented by a benthic piscivore instead of bull trout. Tissue data for this species will be collected in Phase 2.

Table A-4-10. Arsenic dietary toxicity studies for fish

Species	Age/Size at Start of Expt.	Chemical	Exposure	Duration (weeks)	Effect Endpoint	LOEC (mg/kg dw[a])	NOEC (mg/kg dw[a])	Citation	Note
Studies with LOECs									
RBT	29.2 g	sodium arsenite	diet: in prepared food	6	growth: weight gain	30	20	Oladimeji et al. 1984	1
RBT	2 g	disodium arsenate heptahydrate	diet: in prepared food	24	growth: weight gain	33	12	Cockell et al. 1991	2
RBT	7.6 g	disodium arsenate heptahydrate	diet: in prepared food	16	growth: weight gain	44	8	Cockell et al. 1991	3
RBT	48 g	disodium arsenate heptahydrate	diet: in prepared food	24	growth: weight gain	49		Cockell et al. 1991	4
RBT	1.86 g	disodium arsenate heptahydrate	diet: in prepared food	8	growth: weight gain	55		Cockell et al. 1992	5
RBT	35.5 g	disodium arsenate	diet: in prepared food	12	growth: weight gain	58		Cockell and Bettger 1993	3
RBT	13.8 g	disodium arsenate heptahydrate	diet: in prepared food	12	growth: weight gain	60	32	Cockell et al. 1992	3
RBT	3.59 g	disodium arsenate	diet: in prepared food	8	growth: weight gain	120		Cockell and Hilton 1988	3, 6
RBT	3.59 g	arsenic trioxide	diet: in prepared food	8	growth: weight gain	163		Cockell and Hilton 1988	3, 7
Striped bass	juvenile 40g	disodium arsenate heptahydrate	diet: in prepared food	6	growth: weight	188.8	52.3	Blazer et al. 1997	3
Studies with NOECs only									
RBT	4.15 g	dimethylarsenic acid	diet: in prepared food	8	growth: weight gain		1,497	Cockell and Hilton 1988	
RBT	4.15 g	arsenilic acid	diet: in prepared food	8	growth: weight gain		1,503	Cockell and Hilton 1988	

RBT – rainbow trout (*Oncorhynchus mykiss*)

[a] Concentrations are for elemental arsenic. Concentrations are as administered and are assumed to approximate a dry weight concentration.

1) Concentrations in figure and in text in this reference do not agree (20 mg/kg is mentioned both as an effect level and a NOEC in the text); however, it is shown in the figure to be nonsignificant. In this assessment, the 20 mg/kg-diet exposure is thus assumed to be a NOEC. Concentrations are reported as dry weight.
2) Feed consumption did not differ from controls. Body weight gain reduced at 12 weeks, but not at 24 weeks.
3) Effects accompanied by feed refusal.
4) Effects attributed to feed refusal.
5) Growth effects at 60 mg/kg attributed to feed refusal. No feed refusal at 32 mg/kg.
6) Greater than 10% mortality at 500 mg/kg-diet. Fish avoided food at this dose.
7) Greater than 10% mortality at 732 mg/kg-diet. Fish avoided food at this dose.

A.4.2.1.1 Juvenile chinook salmon

In most of the studies reporting dietary arsenic exposure to fish, juvenile fish of the family Salmonidae were used (Table A-4-10). Because rainbow trout are more closely related to chinook salmon than the striped bass also tested, only trout data were used to evaluate effects to juvenile chinook salmon. In total, 9 LOECs and 6 NOECs were identified for trout. The following discussion provides an overview of these studies, and identifies selected NOECs and LOECs for each endpoint.

Survival

Cockell and Hilton (1988) reported increased mortality at dietary arsenic concentrations of 500 mg/kg-diet and 732 mg/kg-diet. However, at these concentrations, fish completely avoided food from the start of the experiment. In this study and the other arsenic studies presented here, feed refusal was not recognized as a toxicological effect. Therefore, mortality was likely attributable to starvation due to reduced food palatability rather than direct toxicity associated with arsenic exposure. No other studies were identified that addressed dietary arsenic effects on fish survival. Therefore, no NOECs and LOECs for survival were identified. It is assumed that NOECs and LOECs identified for growth (a sublethal endpoint) are protective of effects on survival.

Growth

Reduced growth following dietary arsenic exposure was investigated in five studies with rainbow trout (Table A-4-10). In total, 9 LOECs and 6 NOECs were identified for growth. LOECs ranged from 30 mg/kg-diet dosed to 29.2-g rainbow trout (Oladimeji et al. 1984) to 163 mg/kg-diet dosed to 3.59-g rainbow trout (Cockell and Hilton 1988). Oladimeji et al. (1984) reported that juvenile rainbow trout exposed for 2, 4, and 6 weeks to 30 mg/kg-diet had significantly less weight gain than control fish. Fish dosed at 10 mg/kg had no observable effects and those dosed at 20 mg/kg weighed less than controls, but the effect was not statistically significant. The results of Oladimeji et al. (1984) were consistent with studies by Cockell et al. (1991, 1992) using disodium arsenate dosed to juvenile rainbow trout. Cockell et al. (1991) presented results of three studies conducted to differentiate effects on growth from arsenic toxicity from those attributable to reduced palatability of arsenic contaminated food. Results showed that arsenic adversely affected growth at 33 mg/kg-diet with no reduction in feeding at 12 weeks; however, no growth effects were detected at 24 weeks. A more recent study by Cockell et al. (1992) reported a non-significant reduction in weight gain at 32 mg/kg-diet with no effect on feeding. Higher doses resulted in reduced weight gain and reduced feeding. Based on the available data, the Oladimeji et al. (1984) 30 mg/kg-diet LOEC was selected as the LOEC TRV for growth effects.

NOECs ranged from 20 mg/kg-diet dosed to 29.2-g rainbow trout (Oladimeji et al. 1984) to 1,503 mg/kg-diet dosed to 4.15-g rainbow trout (Cockell and Hilton 1988).

The highest NOEC below the selected LOEC (i.e., 30 mg/kg-diet) was 20 mg/kg-diet dosed to 29.2-g rainbow trout for 6 weeks (Oladimeji et al. 1984).

Based on the above analysis and the results presented in Table A-4-10, the NOEC and LOEC selected for growth following dietary arsenic exposure were 20 and 30 mg/kg-diet, respectively.

A.4.2.1.2 Bull trout

In most of the studies reporting dietary arsenic exposure to fish, juvenile fish of the family Salmonidae were used. Because rainbow trout are more closely related to bull trout than the striped bass also tested, only trout data were used to evaluate effects to bull trout. The following discussion provides an overview of these studies and identifies selected NOECs and LOECs for each endpoint.

Survival

No suitable studies were located that address the potential effects of dietary arsenic exposure on survival. NOECs and LOECs identified for growth (a sublethal endpoint) were assumed to be protective of effects on survival.

Growth

Because the most relevant dietary arsenic toxicity studies for bull trout were those discussed above for juvenile chinook salmon, the same NOEC and LOEC for growth were selected. Thus, the NOEC and LOEC selected for bull trout growth following dietary arsenic exposure were 20 and 30 mg/kg-diet, respectively.

Reproduction

No studies were available that addressed potential effects of dietary arsenic exposure on reproduction in fish.

A.4.2.1.3 English sole

This section presents the available dietary arsenic effects data for all fish species tested to assess potential effects on survival, growth, and reproduction to English sole (and other fish represented by this ROC). In total, 10 LOECs and 7 NOECs were identified (Table A-4-10).

Survival

No studies were available that address the potential effects of dietary arsenic exposure on survival. It was assumed that NOECs and LOECs identified for growth (a sublethal endpoint) were protective of effects on survival.

Growth

In addition to the studies reviewed and described above for juvenile chinook salmon, one study was reviewed in which 40-g juvenile striped bass were exposed to disodium arsenate heptahydrate in food (Blazer et al. 1997). However, the NOEC and LOEC from this study were higher than those presented for rainbow trout (Table A-4-10). To

be protective of fish represented by English sole, the lower NOEC and LOEC were selected for English sole (i.e., those selected for juvenile chinook and bull trout). Thus, the NOEC and LOEC selected for growth following dietary arsenic exposure were 20 and 30 mg/kg-diet, respectively.

Reproduction

No studies were available that addressed potential effects of dietary arsenic exposure on reproduction in fish.

A.4.2.2 Copper

Copper, a COPC for all three fish ROCs, was evaluated in this ERA from a dietary perspective (Section A.4.1.2.1). Thus, the literature was searched to identify laboratory studies involving dietary copper exposures associated with survival, growth, and reproductive endpoints. A total of 6 LOECs and 11 NOECs were identified (Table A-4-11). For each ROC, these studies were reviewed to identify a suitable NOEC and LOEC for use in the risk characterization (Section A.7.2).

Table A-4-11. Copper dietary toxicity studies for fish

Species	Age/Size at Start of Expt.	Chemical	Exposure	Duration	Effect Endpoint	LOEC (mg/kg dw[a])	NOEC (mg/kg dw[a])	Citation	Note
Studies with LOECs									
Channel catfish	fingerlings 14.5 g	copper	diet: in prepared food	16 weeks	growth	16	8	Murai et al. 1981	1
Atlantic salmon	fry 0.9 g	copper	diet: in prepared food	3 months	growth	700	500	Lundebye et al. 1999	
RBT	3.8 g	copper sulphate pentahydrate	diet: in prepared food	8 weeks	growth	730	287	Lanno et al. 1985a	2
RBT	3.1 g	copper sulphate pentahydrate	diet: in prepared food	16 weeks	growth	796		Lanno et al. 1985b	3
Atlantic salmon	0.9 g	copper sulphate	diet: in prepared food	3 months	growth	868	638	Berntssen et al. 1999b	
Grey mullet	**9 g**	**copper sulphate pentahydrate**	**diet: in prepared food**	**67 days**	**growth**	**2400**		**Baker et al. 1998**	**7**
Studies with NOECs only									
RBT	138 g	$CuSO_4$	diet: in prepared food	32 days	mortality		200	Handy 1992	
RBT	11.7 g	copper sulphate pentahydrate	diet: in prepared food	24 weeks	growth		664	Lanno et al. 1985a	4
RBT	8 g	copper sulphate	diet: in prepared food	42 days	growth		684	Miller et al. 1993	

Species	Age/Size at Start of Expt.	Chemical	Exposure	Duration	Effect Endpoint	LOEC (mg/kg dw[a])	NOEC (mg/kg dw[a])	Citation	Note
Atlantic salmon	parr 72.2 g	copper sulphate pentahydrate	diet: in prepared food	4 weeks	growth		691.3	Berntssen et al. 1999a	5
RBT	3.8 g	copper sulphate pentahydrate	diet: in prepared food	8 weeks	mortality		730	Lanno et al. 1985a	2
RBT	37 g	copper sulphate pentahydrate	diet: in prepared food	28 days	mortality, growth		1,041.9	Kamunde et al. 2001	6
RBT	130.7 g	$CuSO_4$	diet: in prepared food	28 days	mortality		10,000	Handy 1993	

RBT – rainbow trout (*Oncorhynchus mykiss*)

[a] Concentrations of elemental copper. Except where noted, concentrations were as administered, and were assumed to approximate a dry weight concentration.

1) Significant effects on growth at 4 weeks in 16 and 32 mg/kg treatments. Growth gain per feed consumed was significantly lower for fish fed 8 mg/kg dw.
2) Feed refusal and 19% mortality was observed at 1,585 mg/kg-diet. Concentrations reported as dry weight.
3) Fish were fed on control diet for 12 days after exposure period.
4) Significant effects on growth at 16 weeks. At 24 weeks, growth effects were no longer apparent. Concentrations reported as dry weight.
5) No significant differences in length, weight, or condition factor were observed between the two Cu-supplemented groups and control, but the control showed a significantly higher final weight than starting weight, whereas the Cu-treatments did not.
6) Non-significant growth inhibition at this concentration.
7) Effects associated with reduced feeding.

A.4.2.2.1 Juvenile chinook salmon

Several studies were reviewed that evaluated survival and growth endpoints in fish of the family Salmonidae following dietary copper exposure. Because salmonid species are of the same family as chinook salmon and the two other tested fish species (channel catfish and grey mullet) are not, only salmonid data were used to evaluate effects to juvenile chinook salmon. In total, 4 LOECs and 10 NOECs were reported. The following discussion provides an overview of these studies and identifies selected NOECs and LOECs for each endpoint.

Survival

Four studies were identified that evaluated survival following dietary copper exposure to salmonid species. Only one of the four studies reported reduced survival (though this was not regarded as a LOEC) associated with dietary copper exposure. Lanno et al. (1985a) reported 19% mortality in 3.8 g rainbow trout dosed with 1,585 mg/kg-diet, whereas fish dosed at 730 mg/kg-diet exhibited mortality similar to controls. At 1,585 mg/kg-diet copper, fish actively avoided feeding (attributed to reduced palatability of food); thus, mortality was likely attributable to starvation rather than copper toxicity. Additionally, with increased exposure duration the feed-

to-weight gain ratio of fish fed the high copper diet increased, indicating that they had acclimated to the diet. Three NOECs were identified ranging from 200 mg/kg-diet dosed to 138-g rainbow trout for 32 days (Handy 1992) to 10,000 mg/kg-diet dosed to 131-g rainbow trout for 28 days (Handy 1993). Because no LOEC was available, the highest NOEC may be applicable. However, because toxicity at doses associated with feed refusal is unknown, the results of Lanno et al. (1985a) suggest that adverse effects may occur above 1,585 mg/kg-diet. Based on the above analysis and results presented in Table A-4-11, the NOEC selected for survival following dietary copper exposure was 730 mg/kg-diet based on the NOEC from Lanno et al. (1985a). No LOEC for survival was identified.

Growth

A total of 4 LOECs and 7 NOECs were identified involving potential effects associated with dietary copper exposure on growth of fish of the family Salmonidae. LOECs ranged from 700 mg/kg-diet for 0.9 g Atlantic salmon (Lundebye et al. 1999) to 868 mg/kg-diet for 0.9 g Atlantic salmon exposed over 67 days (Berntssen et al. 1999b). Lundebye et al. (1999) showed growth of 0.9 g Atlantic salmon exposed to copper for a three-month period at dietary concentrations up to 500 mg/kg-diet was not adversely affected, whereas growth of fish exposed to 700, 900, and 1,750 mg/kg-diet was adversely affected. Berntssen et al. (1999a) showed that neither the length, weight, nor condition factor of Atlantic salmon parr fed on 34.2 and 691.3 mg/kg-diet for 4 weeks were significantly different from those variables in controls. However, control fish showed a significantly higher final weight when compared to their initial weight, and copper-dosed fish did not (i.e., change in weight was significant for controls but not for copper-dosed fish). The authors suggested these results indicated adverse effects of copper on growth. However, lack of a significant difference in the final size of fish between the highest dose and the control suggests that these effects were equivocal. Therefore, the selected LOEC was 700 mg/kg from Lundebye et al. (1999). A NOEC of 684 mg/kg was selected at the NOEC based on the Miller et al. (1993) study with 8-g rainbow trout.

Based on the above analysis and results presented in Table A-4-11, the selected NOEC and LOEC for growth following dietary copper exposure were 684 and 700 mg/kg-diet, respectively.

A.4.2.2.2 Bull trout

A total of 4 LOECs and 10 NOECs were identified associated with potential effects from dietary copper exposure to fish of the family Salmonidae. Studies using fish of other families were not included to estimate effects to bull trout because the salmonids tested are more closely related.

Survival

Available studies for the survival endpoint were discussed above for juvenile chinook salmon. Because no additional studies were available that may pertain to bull trout, the same NOEC of 730 mg/kg-diet was selected, and no LOEC was identified.

Growth

Available studies examining effects of dietary exposure to copper on juvenile chinook salmon survival were discussed above. Because no additional studies were available that may pertain to bull trout, the NOEC and LOEC for growth following dietary copper exposure of 684 and 700 mg/kg-diet, respectively, were selected.

Reproduction

No studies were located that address potential effects of dietary copper exposure on reproduction.

A.4.2.2.3 English sole

This section presents the appropriate available data on all fish species tested to assess potential effects on survival, growth, and reproduction to English sole (and fish represented by this ROC) following dietary exposure to copper. In total, 6 LOECs and 11 NOECs were reported (Table A-4-11).

Survival

No additional studies beyond those discussed above under juvenile chinook salmon were identified that address potential effects on survival (Table A-4-11). Because juvenile fish represented by the English sole ROC are present in the LDW, the same NOEC selected for survival of juvenile chinook salmon following dietary copper exposure of 730 mg/kg-diet was also selected for English sole; no LOEC for survival was identified.

Growth

LOECs for growth were reported in six studies ranging from 16 mg/kg-diet for 14.5-g fingerling channel catfish (Murai et al. 1981) to 2,400 mg/kg-diet for gray mullet (Baker et al. 1998). NOECs were reported ranging from 8 mg/kg-diet for fingerling channel catfish (Murai et al. 1981) to 10,000 mg/kg-diet dosed to 103.7-g rainbow trout (Handy 1993). The channel catfish fingerling study resulted in a NOEC and LOEC combination lower than those reported for Atlantic salmon and rainbow trout.

To be protective of the various fish represented by English sole, the lowest available LOEC and corresponding NOEC were selected for English sole (16 and 8 mg/kg-diet, respectively).

Reproduction

No studies were located that address potential effects of dietary copper exposure on reproduction.

A.4.2.3 Mercury

Mercury, a COPC for all three fish ROCs, was evaluated in this ERA from a critical residue perspective (Section A.4.1.1). Thus, the literature was searched to identify laboratory studies reporting whole body tissue residues of mercury associated with survival, growth, and reproductive endpoints. In total, 12 LOECs and 18 NOECs were identified (Table A-4-12). For each ROC, these studies were reviewed to identify a suitable NOEC and LOEC for use in the risk characterization (Section A.7.2.1).

A.4.2.3.1 Juvenile chinook salmon

Almost a third of the studies investigating effects of mercury exposure to fish used juvenile fish of the family Salmonidae as the test species. Because these fish are closely related to chinook salmon, only results from these studies were used to evaluate survival and growth of juvenile chinook salmon in the LDW. A total of 3 LOECs and 8 NOECs were reported in these studies. The following discussion provides an overview of these studies and identifies selected NOECs and LOECs for each endpoint.

Survival

Four studies were reviewed that evaluated potential survival effects on salmonid species following exposure to mercury (Table A-4-12). NOECs for survival were reported in four studies and ranged from 5 µg/g for juvenile rainbow trout exposed via water (Lock 1975) to 29 µg/g for rainbow trout fingerlings exposed to 76.5 mg mercury/kg-diet (Rodgers and Beamish 1982). No LOECs for survival were identified for fish of the family Salmonidae. LOECs for other, less closely related fish, ranged from 0.47 µg/g in mummichog (Matta et al. 2001) to 29 µg/g in medaka (Heisinger and Green 1975). Because these LOECs overlapped with NOECs from more closely related fish, no LOEC was selected. Because no LOEC was identified, no adverse effects are expected at the highest NOEC. Therefore, the highest NOEC is applicable. Thus, the NOEC of 29 µg/g based on results from Rodgers and Beamish (1982) was selected. This NOEC was from a dietary study involving rainbow trout fingerlings similar in size to the chinook salmon as they migrate through the LDW.

Growth

Five studies were identified that evaluated potential growth effects on salmonid species following mercury exposure (Table A-4-12). Rogers and Beamish (1982) exposed 5.5 g hatchery fingerling rainbow trout to methylmercury via contaminated diet. Fish were fed *ad libitum* on diets containing 24.9, 46.9, or 94.8 mg mercury/kg-diet (ww) for 84 days. Weights of fish fed at all doses were significantly lower than controls. The tissue burden associated with the lowest dose was approximately 10 µg/g ww. The authors attribute the observed effects on growth to reduced feeding and reduced digestibility of mercury-contaminated food. As changes in growth were concomitant with changes in food consumption, differences in growth in this experiment may not reflect toxic effects of methylmercury on growth.

Rogers and Beamish (1982), in a separate experiment, further addressed whether effects were due to mercury toxicity or effects on appetite by dosing juvenile rainbow trout in different meal sizes (1% versus 2% body wt/day) at 23.3 and 75.2 mg/kg-diet. Weights of fish fed 1% body wt/day at both doses were not significantly different from controls, whereas those fed 2% body wt/day were significantly different from controls at both doses. However, growth of fish exposed at the lower meal size followed a similar trend to those exposed at 2% body wt/day. Growth effects of the 25 and 75 mg/kg diets at a given meal size were not significantly different, suggesting effects of dietary methylmercury on growth were not directly proportional to quantity of mercury ingested. The authors attributed the observed growth effects to a combination of reduced feeding, reduced food digestibility, and increased metabolism. The results of this set of experiments suggest mercury exposure at all doses tested had potential toxicological effects on growth; however, the toxic effects could not be separated from effects on appetite. Because this study showed toxicity, a whole body tissue residue LOEC of 10 µg/g, associated with the lowest concentration tested in the *ad libitum* diet, was selected as an acceptable LOEC for growth.

Six NOECs were reported ranging from 2.28 µg/g (Phillips and Buhler 1978) to 12 µg/g (Niimi and Lowe-Jinde 1984). Phillips and Buhler (1978) reported that juvenile rainbow trout exposed to mercury in water, or diet, or both water and diet for 24 weeks did not result in impaired growth at body burdens up to 8.63 µg/g, the highest NOEC reported that was less than the selected LOEC of 10 µg/g.

Based on the above analysis and data presented in Table A-4-12, the tissue-residue-based NOEC and LOEC selected for growth effects following mercury exposure were 8.63 µg/g and 10 µg/g, respectively.

Table A-4-12. Mercury whole-body fish tissue residue studies

Test Species	Life Stage	Tissue	Mercury Speciation	Test Conditions	Effect Endpoint	Duration (days)	LOEC (µg/g ww)	NOEC (µg/g ww)	Reference	Notes
Studies with LOECs										
Rainbow trout	embryo-larvae	whole-body: alevins	inorganic mercury	eggs reared in lab on mercury-contaminated sediment 10 days pre to 10 days post hatch	reproduction: egg/embryo survival – reduced 46%	20	0.036		Birge et al. 1977	
Channel catfish	embryo-larvae	whole-body	inorganic mercury	eggs reared in lab on mercury-contaminated sediment	reproduction: egg survival – reduced 49%	10	0.34		Birge et al. 1979	
Mummichog		whole-body	methyl-mercuric chloride	wild male fish exposed to contaminated food @ 0.2, 0.5, 1, 11 µg/g-feed	survival	42	0.47	0.20	Matta et al. 2001	1
Fathead minnow	3 month old	whole-body	mercuric chloride	3 month old fish exposed to 23 to 214 µg/L in flow-through aquaria	growth	60	1.31	0.8	Snarski and Olson 1982	
Fathead minnow	3 month old	whole-body	mercuric chloride	3 month old fish exposed to 23 to 214 µg/L in flow-through aquaria	survival	60	4.18	2.75	Snarski and Olson 1982	
Fathead minnow	larvae-adult	whole-body	mercuric chloride	3 month old fish exposed to 23 to 214 µg/L in flow-through aquaria	reproduction	287	4.47	2.84	Snarski and Olson 1982	
Goldfish	4.5-6.5 cm	whole-body	mercuric chloride	fish exposed to Hg in water	survival	2	5.6		Heisinger et al. 1979	2
Bluegill	juvenile	whole-body	Methyl-mercuric chloride	7-8.5 cm fish exposed to Hg in water	survival	12.5	6.5		Cember et al. 1978	
Brook trout	embryo – adult	whole-body	methyl-mercury	hatchery fish were exposed to 0.03-2.93 µg/L for 39 weeks, progeny were then exposed for 108 weeks	reproduction: reduced egg hatch, reduced juvenile weight	756	9.4	3.4	McKim et al. 1976	5
Rainbow trout	fingerling	whole-body	methyl-mercury	hatchery fingerlings (5.5 g) fed ad-libitum diets containing methylmercury (23.9, 46.9, 94.8 mg/kg-diet ww)	growth	84	10		Rodgers and Beamish 1982	3, 4
Mummichog		whole-body (parental)	methyl-mercuric chloride	parental fish exposed in lab to contaminated food @ 0.2, 0.5, 1, 11 µg/g-feed	F1 fertilization success	42	11	1.1	Matta et al. 2001	
Medaka	embryo	whole-body	mercuric chloride	water exposure; lab	survival	16	29	16	Heisinger and Green 1975	

Test Species	Life Stage	Tissue	Mercury Speciation	Test Conditions	Effect Endpoint	Duration (days)	LOEC (µg/g ww)	NOEC (µg/g ww)	Reference	Notes
Studies with NOECs only										
Guppy	male adult	whole-body	mercuric chloride	fish exposed to 1.023 mg/kg-sediment (dw) mercury in bed sediments in flow-through aquaria	survival	20		0.20	Kudo and Mortimer 1979	
Rainbow trout	fingerling, 3-10 g	whole-body	methyl-mercuric chloride	water exposure	growth	24		2.28	Phillips and Buhler 1978	
Rainbow trout	juvenile	whole-body	methyl-mercury	flow-through juvenile cultured fish (8 g) exposure to 0.25-5 µg/L	survival, growth	84		5.0	Lock 1975	
Rainbow trout	fingerling, 3-10 g	whole-body	methyl-mercuric chloride	water exposure	growth	24		5.67	Phillips and Buhler 1978	
Rainbow trout	fingerling, 3-10 g	whole-body	methyl-mercuric chloride	water exposure	growth	24		8.63	Phillips and Buhler 1978	
Brook trout	embryo – adult	whole-body	methyl-mercury	hatchery fish were exposed to 0.03-2.93 µg/L for 39 weeks	survival, growth	756		9.4	McKim et al. 1976	
Fathead minnow	larvae-adult	whole-body	methyl-mercury	exposed to water concentrations ranging from 0.018 to 0.247 µg/L	survival, growth	336		10.9	Olson et al. 1975	
Rainbow trout	subadult, 100-150 g	whole-body	methyl-mercuric chloride	flow-through juvenile cultured fish exposed to 0.013 or 0.15 µg/L	survival, growth (weight)	75		12	Niimi and Lowe-Jinde 1984	
Mummichog		whole-body (parental)	methyl-mercuric chloride	parental fish exposed in lab to contaminated food @ 0.2, 0.5, 1, 11 µg/g-feed	F1 hatchability, survival, fecundity, F2 larval survival	42		12	Matta et al. 2001	
Mummichog		whole-body	methyl-mercuric chloride	wild fish exposed to contaminated food @ 0.2, 0.5, 1, 11 µg/g-feed	fecundity, fertilization success, offspring weight, female survival	42		12	Matta et al. 2001	6
Rainbow trout	fingerling	whole-body	methyl-mercury	hatchery fingerlings (5.5 g) fed 1% bw/day diets containing methylmercury 23.2 and 76.5 mg/kg-diet ww	survival	84		29	Rodgers and Beamish 1982	

1) Differences were not significant when male and female mortalities were combined.
2) Concentration converted from dry weight to wet weight assuming 20% solids.
3) Reduced growth associated with reduced feeding.
4) Residue approximated from graph.
5) Residue measured in parental fish at 39 weeks.
6) Offspring of medium and high doses were larger than offspring of control fish.

A.4.2.3.2 Bull trout

Bull trout were selected as a representative of piscivorous fish in the LDW for biomagnifying chemicals, such as mercury. Thus, toxicological studies for fish from all families were reviewed for selection of TRVs for bull trout. Several studies were available that addressed potential effects of mercury on survival, growth, and reproduction of fish. In total, 12 LOECs and 18 NOECs were identified for nine species. The following provides an overview of these studies and identifies LOECs and NOECs for each endpoint.

Survival

Eleven studies were reviewed that examined tissue residues and associated potential adverse effects on fish survival following mercury exposure. Five studies reported LOECs associated with decreased survival. LOECs ranged from 0.47 µg/g for mummichog exposed to methylmercury via diet (Matta et al. 2001) to 29 µg/g for medaka embryos exposed to inorganic mercury via water (Heisinger and Green 1975). Matta et al. (2001) exposed wild caught mummichog to methylmercury at 0.50, 1.9, 5.6, and 54 mg/kg-diet over 42 days. Increased mortality was observed in males at body burdens of 0.47 µg/g tissue corresponding to the 1.9 mg/kg-diet dose. No increase in mortality was observed at a tissue residue of 0.2 µg/g corresponding to a 0.50 mg/kg dose. Mortality did not follow a typical dose-response pattern; fish with tissue residues of 1 and 11 µg/g had similar or lower mortality than those with tissue residues of 0.5 µg/g. When male and female mortality were combined, there was no significant difference between mercury-dosed fish and controls. The authors noted that behavior of mercury-dosed male fish was altered such that they became either more aggressive or lethargic, with lethargic fish later dying; such behavioral differences were not observed in female fish. Other authors have observed behavioral alterations indicating respiratory distress (increased respiratory movements) and sluggishness preceding death associated with mercury exposure (e.g., Wobeser 1975; McKim et al. 1976; Rogers and Beamish 1982).

In addition to this study, mercury survival data were available for eight other species, and the next higher survival LOEC (i.e., 4.18 µg/g ww for fathead minnow [Snarski and Olson 1982]) was an order of magnitude higher than the LOEC reported in Matta et al. (2001). Additionally, of the twelve LOECs available for any endpoint, the only lower LOEC were alevin tissue residue values (0.036 and 0.34 µg/g ww in Birge et al. [1977] and Birge et al. [1979], respectively) associated with reproductive effects. Thus, male mummichog survival appears to be highly sensitive to mercury and this TRV is likely to be highly conservative when applied to other species. Based on this analysis and the data presented in Table A-4-12, the LOEC of 0.47 µg/g from Matta et al. (2001) was selected for survival. The NOEC of 0.20 µg/g from the same study was also selected.

Growth

Two LOECs and eight NOECs (tissue residue-based) were identified for growth effects on fish following mercury exposure (Table A-4-12). LOECs ranged from 1.31 µg/g for 3-month-old fathead minnows exposed to mercury in water (Snarski and Olson 1982) to 10 µg/g in rainbow trout exposed through diet (Rodgers and Beamish 1982). NOECs ranged from 0.8 µg/g in three-month-old fathead minnow exposed in water (Snarski and Olson 1982) to 12 µg/g in subadult rainbow trout exposed via water (Niimi and Lowe-Jinde 1984). To be protective of the various fish represented by bull trout, the lower LOEC was selected. The highest NOEC lower than the lowest LOEC was 0.8 µg/g in fathead minnow exposed to mercury in water for 60 days (Snarski and Olson 1982). Note that the selected NOEC and LOEC for the growth endpoint were higher than the selected survival NOEC and LOEC. Generally, effects on sublethal endpoints such as growth are expected to occur at lower concentrations than those resulting in effects on survival. However, no growth effect was observed in Matta et al. (2001), the study from which the survival LOEC/NOEC pair was selected. Additionally, effects on survival were only observed in male fish; no adverse effects were observed on survival or growth of female fish, or male and female fish combined at tissue residues up to 12 µg/g (Matta et al. 2001). Therefore, the available toxicological data for growth were generally consistent with selection of a growth NOEC/LOEC pair higher than the survival NOEC/LOEC pair. Selection of this NOEC/LOEC pair is discussed further in the uncertainty assessment (Section A.7.2.2.3).

Based on the available data presented in Table A-4-12, the tissue residue NOEC and LOEC selected for growth following mercury exposure were 0.8 and 1.31 µg/g, respectively, from Snarski and Olson (1982).

Reproduction

Five studies were identified that evaluated potential effects on fish reproduction associated with whole body tissue residues following mercury exposure. Two studies were reviewed where eggs were reared on mercury-spiked sediments and reduced hatchability and post-hatch survival were observed. Tissue residues were reported for alevins corresponding to a LOEC of 0.036 µg/g for rainbow trout. Controls in this experiment had an alevin tissue concentration of 0.024 µg/g (Birge et al. 1977). In a separate study, the tissue burden reported for embryos associated with a LOEC for catfish embryo was 0.34 µg/g (Birge et al. 1979). Application of these embryo tissue residues to adult fish tissue residues requires a conversion factor. Niimi (1983) suggested that the ratio of mercury in eggs to that in adult fish ranges from 0.04 to 0.10 for rainbow trout, white sucker, white bass, smallmouth bass, and yellow perch. Niimi (1983) suggested that embryo conversion factors should be adjusted two to three times lower to account for water uptake by the egg after spawning. To correct for alevin-to-egg conversion of the data described above, a factor of 3 was applied. These ratios were used with the Birge et al. (1977) embryo tissue residue data to estimate adult female fish tissue residues. As a result, the estimated rainbow trout adult tissue LOEC

calculated with the rainbow trout correction factor (0.017; the egg-to-adult factor of 0.05 divided by 3) was 2.1 µg/g.

Three papers were reviewed that reported reproduction LOECs associated with adult mercury tissue residues. LOECs ranged from 4.47 µg/g for reduced spawning and reduced egg production (Snarski and Olson 1982) to 11 µg/g for reduced fertilization success (Matta et al. 2001). Snarski and Olson (1982) exposed fathead minnows to waterborne mercury for 41 days. Fish with tissue residues of 4.47 µg/g or higher did not mature or spawn. No adverse effects on the number of eggs per female or egg hatchability were apparent at 2.84 µg/g in the adult fish. Similar results were shown by McKim et al. (1976), who exposed brook trout to 0.93 µg/L waterborne mercury for 39 weeks and showed reduced egg hatchability and reduced juvenile weight associated with a parental body burden of 9.4 µg/g. No adverse effects were apparent at a body burden of 3.4 µg/g.

Based on the available data presented in Table A-4-12, the estimated tissue residue selected as the LOEC for reproduction following mercury exposure was 2.1 µg/g based on Birge et al. (1977) with an alevin to adult tissue residue correction factor from Niimi (1983). The NOEC is 0.21 µg/g based on the LOEC/10 (EPA 1997b). Note that there is high uncertainty in these TRVs due to the conversion from alevin to adult tissue residues and in the use of a safety factor of 10 to estimate the NOEC (see Section A.7.2.2).

A.4.2.3.3 English sole

This section presents the available tissue residue data for all fish species tested to assess potential effects on survival, growth, and reproduction to English sole (and fish represented by this ROC) following mercury exposure. The following discussion identifies NOECs and LOECs for each endpoint.

Survival

All relevant studies for English sole were discussed above for bull trout. Therefore, the tissue residue selected NOEC and LOEC for survival were 0.2 µg/g and 0.47 µg/g, respectively.

Growth

All relevant studies for English sole were discussed above for bull trout. Therefore, the tissue residue selected NOEC and LOEC for growth following mercury exposure were 0.8 and 1.31 µg/g, respectively.

Reproduction

All relevant studies for English sole were discussed above for bull trout. Therefore, the tissue residue selected NOEC and LOEC for reproduction following mercury exposure were 0.21 and 2.1 µg/g, respectively.

A.4.2.4 TBT

TBT, a COPC for all three fish ROCs, was evaluated in this ERA from a critical tissue residue perspective (Section A.4.1.1). Thus, the literature was searched for laboratory studies reporting whole body tissue residues of TBT associated with survival, growth, and reproductive endpoints. A total of 5 LOECs and 6 NOECs were identified (Table A-4-13). For each ROC, these studies were reviewed to identify a suitable NOEC and LOEC for use in the risk characterization (Section A.7.2.2).

A.4.2.4.1 Juvenile chinook salmon

One study was identified to assess TBT tissue residues associated with juvenile chinook salmon survival. However, in this study, mean TBT concentrations were only reported in the tissues of the liver (6.4 µg/g ww), brain (1.9 µg/g ww), and muscle (0.298 µg/g ww) in fish that had died following laboratory exposure to TBT. Extrapolation of these tissue data to whole body concentrations is highly uncertain. Therefore, this study was not used in this assessment, and all other available studies relating TBT whole body tissue residues to adverse effects on fish survival or growth were evaluated. This section discusses these studies and identifies NOECs and LOECs for each endpoint.

Survival

Three LOECs and 6 NOECs relating TBT whole-body tissue residues with fish survival were identified (Table A-4-13). LOECs ranged from 3.77 µg/g ww for male guppy exposed to aqueous TBT at concentrations from 8.4 to 1276 µg/L (Tas et al. 1993, 1996) to an LR50-mortality (a statistically derived tissue residue at which 50% mortality occurs) of 8.3 µg/g ww for starry flounder exposed via water (Meador 1997).

Tas et al. (1993) conducted a series of experiments with guppy to determine lethal body burdens of fish exposed to various concentrations of TBT in water. Fish were exposed up to 5 days. Tissue residues associated with mortality increased with exposure concentration. The lowest lethal body burden was in fish exposed for 5 days at 8.4 µg/L (the lowest dose and longest exposure tested). The higher lethal body burdens observed in the higher exposure (46.3 - 1276 µg/L) and shorter duration (45 minutes to 16 hours) tests were attributed to TBT in the whole organism not being at equilibrium with the target organ.[79] . In a 22-day laboratory exposure of starry flounder, Meador (1997) reported a whole-body TBT tissue residue of 8.3 µg/g ww associated with LR50-mortality. Region 10 EPA guidance suggests a safety factor of 50 to derive a NOEC from an LR50 study. Based on this recommendation, a safety factor of 5 was applied to the LR50 to determine a LOEC, resulting in a LOEC of 1.7 µg/g ww (EPA 1997b). This LOEC was selected to represent potential adverse effects to juvenile chinook salmon.

[79] Tas (1996) conducted additional TBT lethal body burden studies. However, in this study, fish were only exposed for 15 minutes. Based on the short exposure duration, this study was not used in this ERA.

Table A-4-13. TBT whole-body tissue residue studies

Test Species	Chemical	Life Stage	Test Conditions	Exposure Route, Concentration	Test Duration (days)[a]	Effect Endpoint	LOEC[b] (µg/g ww)	NOEC[b] (µg/g ww)	Reference	Note
Studies with LOECs										
Goby	Bis[tributyltin] oxide	Male	Lab; flow through	Water; 2 µg/L	84	Decreased gonadal development	1.79		Shimizu and Kimura 1987	
Japanese medaka	Tri-n-butyltin oxide	4 months old 0.3 g	Lab	Diet; 100 mg/kg diet	21	Reproduction: embryo and larval survival	2.39		Nirmala et al. 1999	
Guppy	Tributyltin chloride	Females, 1.16 g	Lab; static	Water; 8.4 µg/L	5.0	Survival	3.77		Tas et al. 1993	1
Starry flounder	Tributyltin	<1 year old	Lab	Water 3 µg/L	22	LR50 survival	8.3		Meador 1997	2
Studies with NOECs										
Guppy	Tributyltin chloride	Female 0.41-0.55 g	Lab; flow-through	Water; 0.28 µg/L	14(14)	Survival		0.07	Tsuda et al. 1990b	3
Guppy	Tributyltin chloride	Female 0.41-0.55 g	Lab; flow-through	Water; 0.54 µg/L	14(14)	Survival		0.26	Tsuda et al. 1990b	3
Carp	Tributyltin chloride	16.5-22.1 g	Lab; flow-through	Water; 1.8 µg/L	14	Survival		2.13	Tsuda et al. 1990a	4
Japanese medaka	Tri-n-butyltin oxide	4 months old 0.3 g	Lab	Diet; 100 mg/kg diet	21	Survival		2.39	Nirmala et al. 1999	
Sheepshead minnow	Bis[tributyltin] oxide	Juvenile	Lab; flow-through	Water; 1.0 µg/L	28	Survival		2.6	Ward et al. 1981	5
Sheepshead minnow	Bis[tributyltin] oxide	Juvenile	Lab; flow-through	Water; 1.61 µg/L	58	Survival		2.95	Ward et al. 1981	5

[a] Additional observation time after exposure is shown in parentheses

[b] Concentration of TBT ion

1) Fish exposed to four 15-minute pulses, each followed by a 24-hr period of elimination. TBT exposure duration appears to have been only 15 minutes.

2) Converted from dry weight based on a reported 17% solids.

3) Residues calculated from bioconcentration factor (BCF) determined in the study.

4) Three studies with pH 6.0 to 7.8. Residues calculated from BCF determined in the study.

5) Radiotracer study; residues measured as tributyltin oxide.

Six NOECs were reported, ranging from 0.07 µg/g ww for guppy exposed to TBT in water for 14 days (Tsuda et al. 1990b) to 2.95 µg/g ww for juvenile sheepshead minnow exposed to TBT in water for 28 days (Ward et al. 1981). Because no NOEC was available below the selected LOEC, based on EPA (1997b) a safety factor of 50 was applied to the LR50 of 8.3 µg/g (Meador 1997) resulting in a selected NOEC of 0.17 µg/g ww.

Based on the above analysis and the results presented in Table A-4-13, the selected NOEC and LOEC for survival of juvenile chinook salmon following TBT exposure were 1.7 and 0.17 µg/g ww, respectively.

Growth

No studies were located that relate whole-body tissue residues to effects on growth of fish.

A.4.2.4.2 Bull trout

No studies reporting TBT whole body tissue residues have been located that assessed the potential effects of TBT on bull trout or fish of the family Salmonidae.[80] Thus, this section presents the available data on all fish species tested to assess potential effects on survival, growth, and reproduction of bull trout following TBT exposure.

Survival

Because the most relevant toxicity studies for bull trout were those discussed above for juvenile chinook salmon, the same NOEC and LOEC for survival were selected. Thus, the tissue residue NOEC and LOEC for survival following TBT exposure were 1.7 and 0.17 µg/g ww, respectively.

Growth

No studies were located that relate whole-body TBT tissue residues to growth effects on fish.

Reproduction

Two studies reported whole-body TBT tissue residues associated with potential reproductive effects. Shimuzu and Kimura (1987) exposed a saltwater goby (*Chasmichthys dolichognathus)* to bis[tributyltin] oxide and observed effects on gonadal development in the male fish. A significant decrease in gonadal development was associated with a whole-body TBT tissue residue of 1.79 µg TBT ion/g ww. Nirmala et al. (1999) exposed Japanese medaka to dietary TBT at 100 mg/kg-diet ww for 3 weeks and showed reduced embryo and larval survival associated with a tissue residue of 2.39 µg/g ww in parental fish.

[80] One study is discussed under the juvenile chinook salmon ROC; however, whole body tissue residues were not reported.

The lower tissue residue LOEC (1.79 µg/g ww) was selected for reproduction based on Shimizu and Kimura (1987). A tissue residue of 0.18 µg/g was estimated as the NOEC based on the LOEC for reproduction divided by a factor of 10 (EPA 1997b).

A.4.2.4.3 English sole

This section presents the available data on all fish species tested to assess potential effects on survival, growth, and reproduction to English sole (and fish represented by this ROC) following TBT exposure. No studies were available for effects on growth.

Survival

Because the most relevant toxicity studies for English sole are those discussed above for juvenile chinook salmon, the same NOEC and LOEC for survival were selected. Thus, the tissue residue NOEC and LOEC selected for survival following TBT exposure were 1.7 and 0.17 µg/g ww, respectively.

Growth

No studies were located that relate whole-body TBT tissue concentrations to effects on growth of fish.

Reproduction

Because the most relevant toxicity studies for English sole were those discussed above for bull trout, the same NOEC and LOEC for reproduction were selected. Thus, the tissue residue NOEC and LOEC selected for reproduction following TBT exposure were 0.18 µg/g ww (estimated based on the LOEC for reproduction divided by a factor of 10) and 1.79 µg/g ww, respectively.

A.4.2.5 DDTs

Total DDTs, a COPC for all three fish ROCs, was evaluated in this ERA from a critical tissue residue perspective. Thus, the literature was searched for laboratory studies reporting tissue residues of these COPCs associated with survival, growth, and reproductive endpoints. Numerous suitable studies were identified involving DDT (Table A-4-14). For each ROC, these studies were reviewed to identify a suitable NOEC and LOEC for use in the risk characterization. A total of 15 LOECs and 16 NOECs were identified.

A.4.2.5.1 Juvenile chinook salmon

Several studies were identified investigating survival and growth endpoints with salmonid species (Table A-4-14). These studies form the basis of the assessment for juvenile chinook salmon. A total of 5 LOECs and 8 NOECs were identified. The following discussion provides an overview of these studies and identifies LOECs and NOECs for each endpoint. As previously discussed in Section A.4.2, the reproduction endpoint was not assessed for chinook salmon.

Table A-4-14. DDT whole-body tissue residue LOEC and NOEC studies

Test Species	Test Duration (days)	Exposure Conditions	Tissue Type	Effect Endpoint	LOEC (µg/g ww)	NOEC (µg/g ww)	Reference	Note
Studies with LOECs								
Brook trout (fry)	156	diet	whole-body	reproduction	3.0		Macek 1968	
Coho salmon (21 month old)	612	water; 0.1–1 µg/L	whole-body	survival	3.0	1.8	Allison et al. 1964	1
Chinook salmon (0.61 g)	40	diet	whole-body	survival	3.65	0.62	Buhler et al. 1969	2, 3
Coho salmon (21 month old)	612	diet; 0.03-1.0 mg/kg-body weight	whole-body	survival	5.5	3.9	Allison et al. 1964	4
Chinook salmon (1.1 g)	40	diet	whole-body	survival	12.1	11.4	Buhler et al. 1969	2, 3
Green sunfish and pumpkinseed	90	field, water (1.02 µg/L)	whole-body	survival	24		Hamelink et al. 1971	2
Mosquito fish	16	water; 4 µg/L	whole-body	survival	26.5		Pillai et al. 1977	2
Fathead minnow (larvae)	5-30	maternal transfer	whole-body (embryo)	reproduction: egg/embryo survival	40.8		Jarvinen et al. 1976, 1977	2
Sailfin molly	21	water; 50 µg/L	whole-body	survival, growth	54.1	30.1	Benton et al. 1994	2, 5
Coho salmon (3.7 g)	60	diet; 100 mg/kg	whole-body	survival	69.6	16.6	Buhler et al. 1969	2, 3
Goldfish (9.1 g)	38	diet and water	whole-body	survival	200		Rhead and Perkins 1984	2, 6, 7
Studies with NOECs only								
Golden shiner	6-15	diet	whole-body	survival		0.025	Courtney and Reed 1971	8
Brook trout (juvenile)	120	diet	whole-body	survival		1.92	Macek and Korn 1970	2
Rainbow trout (15 g)	140	diet; 1.0 mg/kg/wk	whole-body	survival, growth		4.67	Macek et al. 1970	2, 7
Brook trout (fry)	156	diet	whole-body	survival, growth		7.6	Macek 1968	9
Atlantic menhaden	48 (109)[a]	diet; 93 µg/kg	whole-body	growth		24	Warlen et al. 1977	7
Fathead minnow (juvenile-adult)	266	45.6 mg/kg	whole-body	survival		40	Jarvinen et al. 1976, 1977	2
Goldfish (9.1 g)	58	diet and water	whole-body	survival		130	Rhead and Perkins 1984	2, 6, 7

[a] Number in parentheses represents days observed after exposure; the number before represents days over which fish were exposed

1) Tissue residue measured on day 166

2) Residues are sum of detected DDT and its metabolites (DDD and DDE) measured in fish tissue

3) Tissues sampled at 4 and 7 days

4) Control diet was contaminated with 0.76 mg/kg DDT. LOEC tissue residue measured at day 166, NOEC tissue residue measured at day 466. Concentration is total chlorinated hydrocarbons

5) Converted from dry weight to wet weight based on reported 14% solids

6) Converted from dry weight to wet weight assuming 20% solids

7) Radio tracer study

8) Converted from dry weight to wet weight using factor given in paper

9) Length but not weight of male fish was significantly greater than controls

Survival

Four LOECs for survival were identified, ranging from 3.0 to 69.6 µg/g ww (Table A-4-14). The lowest LOEC for a salmonid species was for 21-month-old coho salmon exposed to 0.1 µg/L DDT via water for 312 days. Increased mortality relative to controls from four months through the duration of the study was observed associated with a tissue residue of 3.0 µg/g ww measured 166 days after inception of the experiment (Allison et al. 1964). The highest tissue residue measured in these fish was 1.8 µg/g ww measured at day 466 of exposure. Therefore, a LOEC of 3.0 µg/g ww and a NOEC of 1.8 µg/g ww were selected to represent tissue residue effect level for survival of juvenile chinook salmon.

Growth

The only growth LOEC associated with a whole body DDT tissue residue identified for any fish was reported by Benton et al. (1994). In this study, sailfin molly were exposed to 0 to 100 µg/L o,p'-DDT for 21 days. Percent weight gain for fish exposed to 50 µg/L was significantly lower than control fish, whereas growth of fish exposed to 25 µg/L was not significantly different from controls. These exposures correspond to LOEC and NOEC tissue concentrations of 54.1 and 30.1 µg/g ww DDT, respectively. However, the uncertainty associated with applying these data to juvenile chinook salmon is high in that effects on survival in juvenile chinook occurred at a lower tissue concentration (see above) than survival in sailfin molly (54.1 µg/g), and sublethal effects such as growth are likely to occur at concentrations lower than lethal effects.

Two NOECs for growth were identified for fish of the family Salmonidae. Macek et al. (1970) exposed juvenile rainbow trout to dietary DDT over 168 days and showed growth of fish with tissue residues of 4.67 µg/g ww was not significantly different from control fish. Similar results were found for juvenile brook trout exposed to DDT via the diet over 156 days resulting in a tissue NOEC of 7.6 µg/g ww (Macek 1968). At the highest concentration tested, DDT-exposed fish had significantly greater growth in length than controls. Buhler et al. (1969) also noted apparent increased growth in several species of salmonids dosed orally with DDT. However, the authors attributed this apparent growth increase to size-selective mortality. Positive growth observed by Macek (1968) was not an artifact of size-selective mortality because no mortality occurred in any of the DDT dosed fish. Jarvinen et al. (1977) exposed parental fathead minnows to DDT, as well as eggs and progeny, through diet and water. No effects on growth of progeny at 30 or 60 days after hatching were evident; however, survival was reduced. Based on the above analysis, the growth endpoint does not appear to be a more sensitive endpoint for DDT than survival.

Because no LOEC was identified for growth, no adverse effects are expected at the highest NOEC. Therefore, based on the above information and data presented in Table A-4-14, the tissue residue NOEC selected for growth following DDT exposure was 7.6 µg/g total DDTs. Based on the uncertainty in the available data, no LOEC for growth was selected.

A.4.2.5.2 Bull trout

Bull trout were selected to represent all piscivorous fish in the LDW for biomagnifying chemicals such as DDT. Thus, toxicological studies for fish from all families were reviewed for selection of TRVs for bull trout.

Survival

Nine LOECs and 12 NOECs were located that related DDT tissue residues to potential effects on survival. LOECs ranged from 3.0 µg/g ww for coho salmon exposed via water over 312 days (Allison et al. 1964) to 200 µg/g for 9.1-g goldfish exposed over 38 days via both food and water (Rhead and Perkins 1984). NOECs range from 0.62 µg/g ww for 0.61-g chinook salmon exposed over 40 days via diet (Buhler et al. 1969) to 130 µg/g ww for 9.1-g goldfish exposed over 38 days via both food and water (Rhead and Perkins 1984). To protect the various fish species represented by bull trout, the lowest LOEC and the highest NOEC lower than the LOEC were selected. Therefore, the tissue residue NOEC and LOEC selected for survival of bull trout exposed to DDT were 1.8 and 3.0 µg/g ww, respectively, both from Allison et al. (1969).

Growth

No additional studies beyond those discussed above for juvenile chinook salmon were available. Therefore, the tissue residue NOEC identified for chinook salmon growth (7.6 µg/g ww) was selected for bull trout. Note that the higher NOEC reported for sailfin molly was not selected because the relative sensitivity of the various fish tested is unknown. Based on the uncertainty in the available data, no LOEC was selected. As discussed above for juvenile chinook salmon, growth does not appear to be a more sensitive endpoint than survival for DDT.

Reproduction

Two LOECs[81] and no NOECs were identified for reproduction of fish following DDT exposure. The lowest acceptable LOEC was from Macek (1968). Macek (1968) exposed sexually maturing yearling brook trout for 156 days to dietary DDT at doses of 0.5 to 2.0 mg/kg/wk. The control diet was contaminated with 0.36 mg/kg-diet DDT resulting in a dose of 0.05 mg/kg/week. Mortality of embryos to swim up fry from parental fish with tissue burdens of 3.0 µg/g ww (the lowest tested) was approximately 7%. This was significantly higher than mortality of controls (tissue residues of 0.40 and 0.78 µg/g ww for two control groups). This LOEC relates to a measured embryo concentration of 0.84 µg/g ww.

---

[81] One additional study was identified relating DDT tissue residues in eggs to effects on reproduction of winter flounder. Smith and Cole (1973) exposed wild caught adult winter flounder to DDTs and observed 99% mortality in eggs at egg concentrations of 2.49 µg/g total DDT. No significant effect on egg viability was observed at concentrations of 1.55 µg/g total DDT. However, there was 55% egg mortality in this treatment relative to 52% egg mortality in controls. Control eggs had tissue residues ranging from 0.18 to 1.08 µg/g total DDT. Because of the high control mortality, and control tissue residues similar to those in the treated fish, this study was not considered for selection of TRVs.

Based on this information, the tissue residue NOEC and LOEC selected for reproduction in bull trout following DDT exposure were 0.30 and 3.0 µg/g ww, respectively, based on the LOEC reported in Macek (1968). The NOEC was estimated by dividing the LOEC by 10. There is some uncertainty in this NOEC because the difference in effects between the controls and DDT treated fish were small, and the selected NOEC was lower than the tissue residues reported for controls reported in Macek (1968).

A.4.2.5.3 English sole

This section presents the available data on all fish species tested to assess potential effects on survival, growth, and reproduction to English sole (and fish represented by this ROC) following DDT exposure. A total of 11 LOECs and 13 NOECs were identified (Table A-4-14).

Survival

All available studies were discussed above for bull trout. Therefore, the tissue residue NOEC and LOEC selected for survival of English sole exposed to DDT were 1.8 and 3.0 µg/g ww, respectively.

Growth

No additional studies beyond those discussed above for juvenile chinook salmon were available. Therefore, the tissue residue NOEC identified for chinook salmon growth (7.6 µg/g ww) was selected for English sole. Note that the higher NOEC reported for sailfin molly was not selected because the relative sensitivity of the various fish tested is unknown. Based on the uncertainty in the available data, no LOEC was selected. As discussed above for juvenile chinook salmon, growth does not appear to be a more sensitive endpoint than survival for DDT.

Reproduction

All of the relevant studies addressing potential effects on reproduction following DDT exposure were discussed above for bull trout. Therefore, the tissue residue NOEC and LOEC identified for bull trout (0.30 and 3.0 µg/g ww) were also selected for English sole.

A.4.2.6 PCBs

PCBs, a COPC for all three fish ROCs, were evaluated in this ERA from a critical tissue residue perspective (Section A.4.1.1). Thus, the literature was searched for laboratory studies reporting PCB whole body tissue residues associated with survival, growth, and reproductive endpoints. Table A-4-15 provides an overview of the available PCB laboratory studies and the endpoints of concern. Seventeen LOECs and 18 NOECs were identified. For each ROC, these studies were reviewed to identify a suitable NOEC and LOEC for use in the risk characterization (Section A.7.2).

Table A-4-15. PCB whole-body tissue residue studies

Species	Tissue	PCB Type	Exposure Conditions	LOEC (µg/g ww)	NOEC (µg/g ww)	Effect Endpoint	Reference	Note
Studies with LOECs								
Atlantic salmon	eyed embryos	1:1:1:1 mixture of Aroclors 1016, 1221, 1254, 1260	water exposure of eyed embryos for 48 hours to 6,250 µg/L	1.53		wet weight of alevins 59 days post exposure was significantly reduced	Fisher et al. 1994	1
Rainbow trout	eggs	Aroclor 1254	gravid females fed 200 µg/g for 60 days	1.6		reduced growth of fry	Hendricks et al. 1981	
Sheepshead minnow	whole body	Aroclor 1254	water exposure of parent fish to 0.32 µg/L for 28 days	9.3	1.9	reproduction: decreased fry survival in the first week after hatch	Hansen et al. 1973	
Spot	whole-body	Aroclor 1254	water exposure of 5 µg/L for 20 days	46	27	reduced survival	Hansen et al. 1971	2
Brook trout	fry whole-body	Aroclor 1254	water exposure of eggs to 0.69 to 13 µg/L for 10 days prior to hatch and 118 days after	71	31	reduced growth	Mauck et al. 1978	3,4
Brook trout	eggs	Aroclor 1254	water exposure of 200 µg/L for 21 days	77.9		78% egg hatch compared to 100% in control	Freeman and Idler 1975	5
Pinfish	whole-body	Aroclor 1016	water exposure of 21 µg/L (32 µg/L nominal) for 33 days	106		significant reduction in survival (50% mortality relative to 6% in control)	Hansen et al. 1974	6
Young rainbow trout	whole-body	1:2 ratio of Aroclor 1254:1260	Water exposure of 2.9 µg/L for 90 days	120	70	reduced growth after 90 days exposure	Mayer et al. 1985	
Brook trout	fry whole-body	Aroclor 1254	water exposure of eggs to 3.1 to 13 µg/L for 10 days prior to hatch and 118 days after	125	71	reduced fry survival; 21 to 100% mortality	Mauck et al. 1978	3,7
Sheepshead minnow	fry whole-body	Aroclor 1016	water exposure to 32 µg/L in intermittent-flow toxicity test	200	77	significantly reduced fry survival	Hansen et al. 1975	
Goldfish	whole-body	Clophen A50	water exposure of 500–4,000 µg/L for 5 to 21 days	250		reduced survival	Hattula and Karlog 1972	8
Fathead minnow	terminal residue	Aroclor 1254	PCB exposure in continuous flow aquaria water	458, 361 (female)		reduced spawning, but egg hatchability and fry survival were not affected	Nebeker et al. 1974a,b	
Fingerling coho salmon	whole-body	Aroclor 1254	fed 480 µg/g in diet for 260 days	645	54	reduced survival	Mayer et al. 1977	9

Species	Tissue	PCB Type	Exposure Conditions	LOEC (µg/g ww)	NOEC (µg/g ww)	Effect Endpoint	Reference	Note
Studies or endpoints with NOECs only								
Juvenile chinook salmon	whole-body	Aroclor 1254	fed 17 µg/g in diet for 4 weeks		0.98	No effect on growth, survival, or survival following immunological challenge	Powell et al. (in press)	10
14 -week old Rainbow trout	whole-body	Aroclor 1254	fed 15 µg/g in diet for 32 weeks		8	No effect on growth or survival	Lieb et al. 1974	
mummichog	parental whole-body	Aroclor 1268	adults fed 32 µg/g in diet ~6 weeks; two generations of progeny observed		15	No effects on fertilization success, hatching success, larval survival	Matta et al. 2001	
Juvenile pinfish	whole-body	Aroclor 1254	exposed to 100 µg/L (nominal) for 48 hours		17	No effect on survival	Duke et al. 1970	
Fingerling channel catfish	*whole-body*	Aroclor 1260	*fed 24 µg/g in diet for 193* days		32	No effect on growth or survival	*Mayer et al.* 1977	
Juvenile chinook salmon	whole-body	Aroclor 1254	oral gavage of 1,000 µg/g fish		60	No effect on survival	Powell et al. *(in press)*	11
Sheepshead minnow	fry whole-body	Aroclor 1016	exposed to 32 µg/L in intermittent-flow toxicity test		77	No effect on fertilization success, survival of embryos to hatching, or survival of fry for 2 weeks	Hansen et al. 1975	
Young rainbow trout	whole-body	1:2 ratio of Aroclor 1254:1260	water exposure of 2.9 µg/L for 90 days		120	No effect on survival, or survival following immunological challenge	Mayer et al. 1985	12

1) No significant differences were observed in hatching success or survival. PCBs dissolved in dimethyl sulfoxide (DMSO). Controls exposed to 0.5% DMSO.
2) Note that mortality did not appear directly related to the body burden, rather the body burden increased with exposure duration. Although mortality of spot (51 to 62%) was similar in three tests of different duration (20 to 45 days), the body burden (46 to 152 µg/g) increased with exposure duration.
3) Tissue residue measured at 118 days.
4) Growth effect reported at 48 days but disappeared at 118 days.
5) Note that none of the eggs hatched in water containing 200 µg/L PCBs and 3.8 mg/L Corexit 7664, which was present in a control and the PCB exposure tank.
6) In a 42-day exposure, 13 µg/L measured in water resulted in 44% mortality and a body burden of 620 µg/g, whereas an 18-day exposure of 59 µg/L resulted in 50% mortality and a body burden of 205 µg/g.
7) Median hatching time and egg hatchability were not affected. Larval growth was initially reduced, but not by the end of the test at 118 days.
8) Reported PCB concentrations are lethal body burdens. PCBs dissolved in acetone (0.5 mL/L). Controls exposed to 0.5 mL/L acetone.
9) Fish with 645 µg/g began dying at day 260 and all were dead within 5 days.
10) Fish were exposed to doses from 0.43 to 17 µg/g-diet Aroclor 1254 for 4 weeks in spiked feed followed by 13 weeks of unspiked feed during growth measurement and immunological testing. Body burdens in the high dose ranged from 0.74 to 0.98 µg/g over the 13-week period.
11) Treatment groups were exposed to a range of 10 to 1,000 µg/g Aroclor 1254 by oral gavage and monitored for 96 hours.
12) There was a trend toward increased mortality although it was not significantly different.

While a large body of literature is available on this topic, this section focuses on studies involving Aroclors (specific congener mixtures that were used in industrial applications) rather than individual congeners. Potential effects associated with exposure to individual congeners are discussed in the uncertainty assessment (Section A.7.2.2).

A.4.2.6.1 Juvenile chinook salmon

Several PCB studies are available involving relevant endpoints and salmonid species (Table A-4-15). These studies are discussed below.

Survival

Studies in Table A-4-15 reporting reductions in survival following exposure to PCBs are discussed in two categories: 1) those without immunological challenge, and 2) those resulting in reduced survival following immunological challenge (i.e., exposure to a pathogen following exposure to PCBs and subsequent monitoring of survival). The second endpoint is discussed for juvenile chinook salmon, but not the other two fish ROCs, because a study conducted with juvenile chinook salmon collected from the LDW suggested that these fish may experience increased mortality due to reduced immunocompetence (Arkoosh et al. 1998b). This study is discussed in Section A.4.3. Additional studies suggested that PCBs and PAHs may be the causative agent (Varanasi et al. 1993; Arkoosh et al. 1998a). Therefore, this endpoint was only evaluated for PCBs (below) and PAHs (Section A.4.2.7.1).

Survival Following PCB Exposure

One relevant study was identified that reported a LOEC for survival of a salmonid species. Mayer et al. (1977) exposed fingerling coho salmon to PCBs via diet for 260 days and observed decreased survival at a tissue residue of 645 µg/g ww.

NOECs for survival were reported in four studies and ranged from 0.98 µg/g ww for juvenile chinook salmon exposed to Aroclor 1254 via diet (Powell et al. in press) to 120 µg/g ww for 17-day-old rainbow trout exposed to 2.9 µg/L of a 1:2 Aroclor 1254:1260 mixture for 90 days (Mayer et al. 1985). The highest NOEC less than the lowest LOEC was 120 µg/g from Mayer et al. (1985).

The LOEC of 645 µg/g ww from Mayer et al. (1977) was selected to represent potential effects on survival of juvenile chinook salmon following PCB exposure. The selected NOEC was 120 µg/g ww (Mayer et al. 1985).

Survival Following PCB Exposure and Subsequent Immunological Challenge

Two studies were identified that evaluated survival of *Oncorhynchus* species following exposure to PCBs and subsequent immunological challenge. Powell et al. (in press) reported no reduction in juvenile chinook salmon survival relative to controls following a dietary exposure of up to 17 µg/g ww Aroclor 1254 and subsequent 14-day challenge with *Vibrio anguillarum* in fresh water. Tissue concentrations of

Aroclor 1254 in juvenile chinook salmon were approximately 0.98 µg/g ww for the highest dose over the duration of the primary immunocompetence study.

Mayer et al. (1985) exposed 18-day-old rainbow trout (0.25 g) to a 1:2 ratio of Aroclor 1254:1260 at water concentrations up to 2.9 µg/L for 90 days. Subsamples of fish were exposed to an LD50 concentration of *Yersinia ruckeri*, the bacterium causing enteric redmouth disease, via flush exposure at several time points in the PCB exposure. Separate disease challenge trials were conducted at days 45, 60, and 90 of PCB exposure and again at 30 and 60 days following PCB exposure. In all exposures, time to 50% mortality was not significantly different between PCB-dosed fish and controls. The authors reported a trend toward higher resistance to disease in those fish exposed to PCBs. The body burden associated with the highest NOEC was 120 µg/g ww. In a separate experiment reported in the same paper, juvenile rainbow trout were exposed to the PCB mixture at 1.5 µg/L for 60 days, and then dosed once with an LD50 concentration of enteric redmouth disease via intraperitoneal injection or via flush exposure. Mortalities were recorded over 10 days. Again, there was no significant difference in survival between PCB-dosed fish and controls for either of the pathogen exposure routes.

These results are consistent with those of Snarski and Olson (1982). This study found that rainbow trout exposed to PCBs (Aroclor 1254) at 14.7 µg/L for 30 days were significantly less susceptible to the bacteria *Aeromonas hydrophila* than were controls. No tissue burdens were reported in this study, however.

Based on the results of the above review, the NOEC selected for survival following PCB exposure and subsequent immunological challenge was 120 µg/g ww. No LOEC was identified.

Growth

The only suitable LOEC found for juvenile salmonid species was 120 µg/g ww in juvenile rainbow trout following a 90-day exposure to a 1:2 ratio of Aroclor 1254:1260 at 2.9 µg/L (Mayer et al. 1985). No effects on growth were observed in this study at a tissue residue of 70 µg/g ww. NOECs from studies of salmonid species ranged from 0.98 µg/g ww in juvenile chinook salmon following a four week dietary exposure (Powell et al. in press) to 70 µg/g ww in juvenile rainbow trout following water exposure for 90 days (Mayer et al. 1985).

Based on the results summarized above, the NOEC and LOEC selected for growth effects following PCB exposure were 70 and 120 µg/g ww, both from Mayer et al. (1985).

A.4.2.6.2 Bull trout

Bull trout were selected to represent all piscivorous fish in the LDW for biomagnifying chemicals such as PCBs. Thus, toxicological studies for fish from all families were reviewed for selection of TRVs for bull trout. Several studies were identified that

assessed potential effects of PCBs on survival, growth or reproduction in fish species (Table A-4-15). The selection of NOECs and LOECs for bull trout is discussed below.

Survival

Reduced survival following PCB exposure was reported in six studies (Table A-4-15). LOECs ranged from 46 µg/g ww in whole-body spot following an Aroclor 1254 water exposure for 20 days (Hansen et al. 1971) to 645 µg/g ww in whole-body fingerling coho salmon following dietary exposure (Mayer et al. 1977).

NOECs were reported in eight papers, and ranged from 0.98 µg/g ww in juvenile chinook salmon following dietary exposure (Powell et al. in press) to 120 µg/g ww in rainbow trout following a 90 day water exposure (Mayer et al. 1985). The highest NOEC below the lowest LOEC was 27 µg/g ww (Hansen et al. 1971).

Based on the above analysis and the results represented in Table A-4-15, the bull trout NOEC and LOEC selected for survival following PCB exposure were 27 and 46 µg/g ww, respectively, both from Hansen et al. (1971).

Growth

Four growth-related LOECs were identified ranging from 1.53 µg/g ww in Atlantic salmon fry exposed as eyed embryos to PCBs via water for 48 hours (Fisher et al. 1994) to 120 µg/g ww in young rainbow trout following a 90-day water exposure (Mayer et al. 1985). Hendricks et al. (1981) reported similar results to Fisher et al. (1994). Hendricks et al. (1981) reported reduced growth of fry from gravid female rainbow trout exposed for 60 days to 200 µg/g ww PCBs via diet at a tissue burden of 1.64 µg/g measured in eggs. Fish represented by the bull trout ROC can be exposed as eyed embryos within the LDW, so studies with early life stages are relevant to the bull trout ROC even though bull trout do not spawn in the LDW and their embryos are not directly exposed to LDW sediment-associated chemicals. However, because no egg or embryo tissue residues have been collected from the LDW, this LOEC could not be directly compared with available data. The rainbow trout LOEC was selected because a rainbow trout egg-to-adult tissue residue conversion was available. Thus, a LOEC for rainbow trout was estimated with less uncertainty than for Atlantic salmon.

Niimi (1983) showed that adult tissue burdens can be estimated based on egg or embryo concentrations.[82] Niimi (1983) determined the total PCBs egg:adult factor for rainbow trout to be 0.43. Therefore, the adult tissue concentration was estimated by dividing the egg LOEC of 1.6 µg/g ww by 0.43, resulting in an estimated LOEC of 3.72 µg/g ww in adult tissue.

Five growth-related NOECs were identified ranging from 0.98 µg/g ww in juvenile chinook salmon following a four week dietary exposure (Powell et al. in press) to 70 µg/g ww in juvenile rainbow trout following a 90 day exposure to a 1:2 ratio of

[82] Results from Niimi (1983) were also used to predict adult tissue concentrations based on egg concentrations in the Sheboygan ecological risk assessment (EVS and NOAA 1998).

Aroclor 1254:1260 at 2.9 µg/L for 90 days (Mayer et al. 1985). The highest NOEC below the selected LOEC was 0.98 µg/g ww.

The LOEC selected was 3.72 µg/g ww based on Hendricks et al. (1981) and applying the Niimi (1983) conversion factor. Use of this LOEC in the risk characterization is uncertain, however, because of the uncertainty associated with application of egg:adult correction factor. The highest NOEC below the selected LOEC was 0.98 µg/g ww (Powell et al. in press).

Reproduction

A small number of studies were available on potential reproductive effects in fish following laboratory exposures to PCBs (Table A-4-15). Three studies reported LOECs associated with endpoints ranging from egg hatching and spawning success to larval survival. Hansen et al. (1973) reported reduced survival of sheepshead minnow fry one week after hatch following water exposure of Aroclor 1254 to parent fish. The tissue burden LOEC in adult females was 9.3 µg/g ww; fry of adult females with a tissue burden of 1.9 µg/g ww did not have reduced survival.

NOECs were reported in four studies and ranged from 1.9 µg/g ww described above (Hansen et al. 1973) to 77 µg/g ww measured in sheepshead minnow fry exposed via water (Hansen et al. 1975).

Based on the above analysis, the tissue residue NOEC and LOEC selected for bull trout reproduction following PCB exposure were 1.9 and 9.3 µg/g ww, respectively, both from Hansen et al. (1973).

A.4.2.6.3 English sole

This section presents the available data for all fish species tested to assess potential effects on survival, growth, and reproduction to English sole (and other fish represented by this ROC) following exposure to PCBs.

Survival

All available studies were discussed above for bull trout. Therefore, the tissue residue NOEC and LOEC selected for survival of English sole exposed to PCBs were 27 and 46 µg/g ww, respectively.

Growth

All available studies were discussed above for bull trout. Therefore, the tissue residue NOEC and LOEC selected for growth of English sole exposed to PCBs were 0.98 and 3.72 µg/g ww, respectively.

Reproduction

All of the relevant studies addressing potential effects on reproduction following PCB exposure were discussed above for bull trout. Therefore, the tissue residue NOEC and LOEC selected for reproduction of English sole exposed to PCBs were 1.9 and 9.3 µg/g ww, respectively.

A.4.2.7 PAHs

PAHs were selected as a COPC for juvenile chinook salmon and English sole and were evaluated from a dietary perspective. In this section, studies involving dietary exposure of fish to PAHs were reviewed for potential effects on survival, growth, and reproduction. Because fish rapidly metabolize PAHs (Varanasi 1989), the critical residue approach was not applicable.

For the purposes of this ERA, only studies in which effects were measured following a dietary PAH exposure were considered. Studies in which fish were exposed to PAH via IP injection are discussed when there were no dietary studies available for the endpoint. However, as previously discussed in Section A.4.2, the IP study results were not be utilized in the ERA to determine a NOEC or LOEC because the injected dose cannot be related to the environmental exposure of the fish.

Fish are exposed to PAH mixtures in the LDW. Studies identified have exposed fish to either a model PAH mixture or to the PAH compound benzo[a]pyrene (BaP). For each ROC, these studies are reviewed to identify a suitable dietary NOEC and LOEC to use in the risk characterization (Section A.7.2.1).

A.4.2.7.1 Juvenile chinook salmon

No studies with PAH mixtures were identified for juvenile chinook salmon or any related species evaluating survival or growth endpoints. Thus, the literature was searched for studies involving exposure to individual PAHs. Two BaP studies were identified that evaluated survival and growth endpoints in salmonids (Table A-4-16). These studies are discussed below.

Table A-4-16. PAH dietary toxicity studies for fish

Test Species	Age/Size of Fish	Test Duration (days)	Study Conditions	Effect Endpoint	LOEC (mg/kg)	NOEC (mg/kg)	Reference	Notes
Rainbow trout	2 in.	28 days	Prepared diet	Growth	1,000	100	Hart and Heddle 1991	1
Rainbow trout	3 mo. old, 3.3 g	18 months	Prepared diet	Growth (reduced body weight)	1,000		Hendricks et al. 1985	2, 3
Rainbow trout	3 mo. old, 3.3 g	18 months	Prepared diet	Survival		1,000	Hendricks et al. 1985	2, 3
English sole	Juvenile (40-55 mm)	10-12 days	Diet of polychaetes exposed for 28 days	Growth	116	47	Rice et al. 2000	4

Note: All studies were conducted using the single PAH benzo(a)pyrene (BAP)

1) Dose was as administered in commercial fish food. Dry weight is assumed (see text).

2) Dose was in Casein diet, wet weight is assumed (see text).

3) Total mortality was higher in the control group. This paper also reported that a nine-week study using 500 and 1,000 mg PAH/kg diet (ww) was conducted to determine a suitable dose for the 18 month study. Results on growth were not presented but the authors state that the fish tolerated both the 500 mg/kg and the 1,000 mg/kg doses well.

4) Dose presented as concentration in prey tissue (dry weight).

Survival

One study was identified that presented a NOEC for survival of salmonid species following dietary BaP exposure. Hendricks et al. (1985) exposed 3.3-g juvenile rainbow trout to 1,000 mg/kg BaP via diet for 18 months. Total mortality was higher in the controls than in the BaP-exposed fish. This paper did not indicate whether dietary concentrations were wet or dry weight. However, the diet was mixed using an electric drill with a mixing whip, and is thus likely to be somewhat liquid. It was assumed the food had a moisture content similar to juvenile chinook salmon diet for direct comparison to wet weight exposure data.

Based on the results of this study, a NOEC of 1,000 µg/g-diet ww was selected to represent survival of juvenile chinook salmon following exposure to PAHs. Use of BaP as a surrogate for total PAHs is addressed in the uncertainty assessment (Section A.7.2.2). No LOEC was identified.

Survival Following Immunological Challenge

Two studies were identified (but not presented in the table, as discussed below) in which juvenile chinook salmon were exposed to PAH mixtures, and survival following exposure to a pathogen was assessed (Arkoosh et al. 1998a; Palm et al. in prep). Arkoosh et al. (1998a) dosed fish via IP injection, an exposure mechanism that cannot be related to an environmental concentration. Nevertheless, Arkoosh et al. (1998a) is reviewed here to assess the potential ability of PAHs to cause increased mortality as a result of suppressed immunocompetence, but no NOEC or LOEC relevant to natural exposure regimes could be calculated because of the IP injection-dosing mechanism used (see Section A.4.2).

Arkoosh et al. (1998a) exposed hatchery-reared juvenile chinook salmon to either a model mixture of 10 high-molecular weight PAHs[83] at a dose equivalent to 6.3 mg total PAH/kg-fish ww or a control (acetone-emulphor carrier) via intraperitoneal injection. Seven days after PAH injection, these fish were exposed to an LC50 dose of the bacterial pathogen, *Listonella anguillarum* (formerly *Vibrio anguillarum*). Mortality was compared among treatments. The net cumulative mortality was reported to be significantly higher in the PAH-exposed fish at 7 days after the bacterial challenge. However, significant mortality occurred in PAH treatments that was not attributable to *Listonella* (36% at day 7, 46% at day 14). Specifically, when one out of every three mortalities were necropsied for the presence of *L. anguillarum*, 36% at day 4 and 46% at day 7 did not test positive for *L. anguillarum*. Because not all mortalities were examined for presence of *L. anguillarum*, the actual portion of the mortalities caused by reduced immunocompetence cannot be accurately determined. Based on the above analysis, it is not clear if this PAH mixture is capable of causing increased mortality as a result of immunosuppression.

---

[83] PAHs injected were fluoranthene, pyrene, benz[a]anthracene, chrysene, benzo[b]fluoranthene, benzo[k]fluoranthene, BAP, indeno[1,2,3-cd]pyrene, dibenz[a,h]anthracene, benzo[ghi]perylene. Other treatments included two Hylebos waterway sediment extracts and hexachlorobutadiene.

Additional studies have been conducted in which juvenile chinook salmon were exposed to PAH mixtures through a dietary pathway at concentrations designed to bracket dietary exposure in the LDW (Palm et al. in prep). These studies were conducted to investigate potential effects of PAHs through an environmentally relevant exposure regime (i.e., through diet rather than IP injection). Following a 28-day exposure, juvenile chinook salmon were challenged with *Listonella anguillarum*, and mortality was monitored for a 14-day period. Results from these studies are currently being analyzed, but will be available for use in the Phase 2 ERA.

Growth

Two studies were identified that presented LOECs for growth of salmonid species (rainbow trout) following dietary BaP exposure (Table A-4-16). Hendricks et al. (1985) exposed 3.3-g juvenile rainbow trout to 1,000 mg BaP/kg diet for 18 months. This paper did not indicate whether dietary concentrations presented were based on wet or dry weight. However, the diet was mixed using an electric drill with a mixing whip and was thus likely to be somewhat liquid, so it was assumed the food had moisture content similar to juvenile chinook salmon diet for direct comparison to wet weight exposure data. Growth of BaP-exposed fish was significantly less than that of control fish. Hart and Heddle (1991) exposed juvenile rainbow trout to 10, 100, and 1,000 mg BaP/kg-diet for 28 days. Weight basis of dietary PAH concentration was not presented. The diet was commercial fish food, which was likely to have low moisture content, so a dry weight concentration was assumed (a conservative assumption). Fish exposed at 1,000 mg/kg-diet had significantly lower growth than controls, whereas fish exposed at the lower doses did not.

Based on this analysis, the NOEC and LOEC for growth of juvenile chinook salmon following exposure to PAHs are 100 mg/kg-diet (dw) and 1,000 mg/kg-diet (dw), respectively. Uncertainties associated with the use of BaP as a surrogate for total PAHs are addressed in the uncertainty analysis (Section A.7.2.2).

Additional studies have been conducted in which juvenile chinook salmon were exposed to a relevant PAH mixture through a dietary pathway at concentrations designed to bracket dietary exposure in the LDW (Palm et al. in prep). Growth of juvenile chinook salmon following PAH exposure was assessed over a several month period. Results from this study are currently being analyzed, and will be available for the Phase 2 ERA.

A.4.2.7.2 Bull trout

As previously discussed, PAHs were not selected as a COPC for bull trout.

A.4.2.7.3 English sole

This section presents the available data for all fish species tested to assess potential effects on survival, growth, and reproduction to English sole (and other fish represented by this ROC) following exposure to PAHs. No dietary studies associated with exposure to PAH mixtures and potential effects on survival, growth, or

reproductive endpoints were identified. Thus, the literature was searched for studies involving individual PAHs. Three LOECs and four NOECs were identified (Table A-4-16). These studies are discussed below.

Survival

One study was identified that presented a NOEC for survival of rainbow trout following dietary BaP exposure. Hendricks et al. (1985) exposed 3.3-g juvenile rainbow trout to 1,000 µg/g BaP via diet for 18 months. Results show that total mortalities were higher in the controls than in the BaP-exposed fish.

Based on the results of this study, a NOEC of 1,000 µg/g-diet (assumed ww) was selected to represent survival of English sole following exposure to PAHs. No LOEC was identified.

In addition to direct mortality, indirect mortality as a result of liver lesions potentially caused by PAH exposure was assessed for English sole in Puget Sound. The most direct and widely cited study suggesting a link between PAH exposure and liver lesions in English sole was Schiewe et al. (1991). In this study, 2-year-old sole collected from Useless Bay (a non-urban site in northern Puget Sound) were injected every 4 weeks over 1 year with 12 mg/kg body weight BaP. After injection, fish were held for 6 months before they were sacrificed and their livers examined. BaP-exposed fish had elevated prevalences of preneoplastic lesions. None of the control fish exhibited these lesions. Survival in the PAH-dosed fish was similar to that among the controls. No correlation between the presence of lesions and adverse effects on survival was found in this study.

Growth

LOECs were presented in three studies ranging from 116 mg/kg diet dw for English sole (Rice et al. 2000) to 1,000 mg/kg diet dw for rainbow trout (Hart and Heddle 1991; Hendricks et al. 1985). Rice et al. (2000) evaluated growth in juvenile English sole following dietary exposure to BaP via a trophic transfer study using juvenile English sole (40-55 mm). In this study, the polychaete *Armandia brevis* was exposed to BaP-spiked sediment. After 28 days, the exposed polychaetes were then fed to the sole. A significant reduction in growth was reported for BaP-exposed fish (growth rate of 0.4% ww per day) relative to control fish (growth rate of 1.1% ww per day) (Table A-4-16). The dietary-based NOEC was 47 mg/kg dw in polychaete tissue and the LOEC was 116 mg/kg dw in polychaete tissue. The exposure of the fish was quantified in terms of the measured PAH concentration in polychaete tissue and sediment. The actual dose can be calculated from the feeding rates, which ranged from 5.4% to 7% initial fish body wet weight per day. It should be noted that there was variability in feeding rates associated with the initial study that resulted in the LOEC concentration. Reduced growth was found in the three treatment groups with feeding rates lower than the control fish feeding rate. The authors concluded that the reduced growth was attributable to chemical exposure and not the reduced feeding rate.

In another dietary study, effects of BaP exposure to adult female croaker, no effect on fish growth was found at a dose of 5.7 µg/g fish/day (Thomas 1988). Neither the PAH concentrations in the experimental diet nor the feeding rate were reported. Therefore, this growth NOEC could not be related to exposure data in the risk characterization, and was not presented in the table.

Based on the available data, the dietary NOEC and LOEC presented in Rice et al. (2000) was used to evaluate growth effects in English sole (i.e., 47 mg/kg-diet and 116 mg/kg-diet, respectively). Uncertainty associated with the use of BaP as a surrogate for total PAHs is discussed in the uncertainty assessment (Section A.7.2.2).

Reproduction

No studies were identified in which reproductive effects were associated with dietary exposure of fish to individual PAHs or mixtures. However, in a study of the effects on adult female croaker associated with dietary exposure to BaP (Thomas 1988), reduced levels of 17 beta-estradiol and reduced ovarian growth were associated with a dose of 5.7 mg BaP/kg fish/day. These results suggest that BaP exposure can affect fish in ways that may alter reproduction (Thomas 1988). However, it is not known whether these effects would result in reduced fecundity or offspring viability. Additionally, as noted under the growth endpoint, neither the PAH concentrations in the experimental diet nor the feeding rate were reported. Therefore, data from this study could not be related to exposure data in the risk characterization, so no NOEC or LOEC for English sole using reproduction as an endpoint was available.

A.4.3 Regional Field Studies

Numerous regionally relevant studies have been conducted with either field-collected juvenile chinook salmon and English sole, fish exposed to field-collected sediments in the laboratory, or fish exposed through IP injection of sediment extracts from the Hylebos Waterway (Commencement Bay, Tacoma, Washington) or Eagle Harbor. These studies are presented separately from effects studies in Section A.4.2 because in these studies juvenile chinook salmon or English sole were exposed to a mixture of chemicals, and thus assessing potential chemical-specific cause and effect was less straightforward.

Studies included in this section fall into one of three categories: 1) studies involving LDW-exposed fish; 2) studies in which fish were exposed through IP injection of extracts from sediments from other locations; or 3) studies in which fish were exposed to field-collected sediments in the laboratory. Studies with LDW-exposed fish are discussed in detail in this section due to the relevance of the exposure route (field exposure) and mixture of chemicals. However, as previously indicated, chemical-specific NOECs and LOECs cannot be determined from these studies because the fish were exposed to chemical mixtures under uncontrolled conditions. The studies involving IP injection of the Hylebos Waterway and Eagle Harbor sediment extracts and lab exposures to field-collected sediment are acknowledged in this section because

they involved juvenile chinook salmon and English sole and may contribute to the weight-of-evidence analysis in the ERA. However, these studies are not discussed in detail because fish were exposed to chemical mixtures that are not specific to the LDW, and the exposure route is not ecologically relevant.

A.4.3.1 *Juvenile chinook salmon*

Studies conducted with juvenile chinook salmon exposed in the LDW or through IP injection of Hylebos Waterway sediment extracts are summarized in Table A-4-17. The studies involving LDW-exposed fish are discussed below under survival and growth endpoints. Included in the survival endpoint for juvenile chinook salmon are studies examining immunocompetence with survival as the measurement endpoint (i.e., exposure to a pathogen following exposure to chemical mixtures and subsequent monitoring of survival). As previously discussed, this endpoint is discussed for juvenile chinook salmon (and not English sole) because a study conducted with juvenile chinook salmon collected from the LDW suggested that these fish may experience increased mortality due to reduced immunocompetence (Arkoosh et al. 1998b).

Table A-4-17. LDW-specific and regional laboratory studies of juvenile chinook salmon survival, immunocompetence, and growth

Study	Exposure	Effects Endpoints
LDW-specific studies		
Varanasi et al. 1993	LDW field/ GR hatchery	Survival, growth
Arkoosh et al. 1998b	LDW field/ GR hatchery	Survival-disease susceptibility (*L. anguillarum*)
Casillas et al. 1995a,b	LDW field/ GR hatchery	Growth, insulin-like growth factor
Regional studies		
Hylebos Round II part 1(Arkoosh et al. 1998a)	IP injection of HWSE	Survival-disease susceptibility (*L. anguillarum*)
Hylebos Round II part 2 (Casillas et al. 1998a)	IP injection of HWSE	Growth, survival

GR – Green River

HWSE – Hylebos Waterway sediment extract

In the Hylebos Round II fish injury studies, juvenile chinook salmon were injected with two extracts made from Hylebos Waterway (in Commencement Bay) sediments. In addition to other chemicals, the extracts contained total PAHs, PCBs, and hexachlorobutadiene. Control fish were dosed with a reference sediment extract from Nisqually estuary sediments. Following injection, fish were examined for differences in survival, growth, biomarkers, and disease susceptibility (Casillas et al. 1998a; Arkoosh et al. 1998a). As noted above, these studies cannot be used in the ERA because it is not possible to quantitatively translate IP injection doses to ecologically relevant doses, such as dietary doses.

A.4.3.1.1 Survival

This section discusses the only study available that investigated potential effects of LDW exposure on survival of juvenile chinook salmon. Varanasi et al. (1993) collected juvenile chinook salmon from Puget Sound urban estuaries and hatcheries located upstream of each estuary. The fish were to be held in the laboratory for 120 days, and survival and cumulative growth within and between river systems was compared. The Nisqually estuary and upstream hatchery served as a non-urbanized control estuary/river system. Juvenile chinook salmon were collected from hatcheries and the respective estuaries of the Green/Duwamish, Puyallup, and Nisqually rivers in 1990. In 1991, fish were sampled only from the Green River hatchery and the LDW near Kellogg Island. Hatchery fish were collected just prior to release and estuary fish were collected approximately 2 weeks later. Whole fish and liver tissues, stomach contents, blood and bile were sampled for organic chemical and biomarker analyses. In 1990, the experiment was terminated early, after 40 days, because of a high incidence of bloating in all groups of unknown etiology. Results show that at test termination, survival of fish from the LDW (56%) and the Puyallup estuary (58%) was significantly lower than survival of Green River hatchery fish (86%). Survival of Nisqually estuary fish (81%) was not statistically different from that of upstream Kalama Creek hatchery fish (88%).

In 1991, the study was repeated with only fish from the LDW and the Green River hatchery. After changes in husbandry practices (i.e., reduced light, minimized handling, different anesthetic, reduced feeding), the study was terminated after 84 days because of high mortality of undetermined cause in all groups. At test termination, survival was significantly lower for the LDW-collected fish (59%) relative to Green River hatchery fish (77%). Due to the study design and high control mortality, it was not possible to determine which of the various potential factors or combination of factors could have caused this difference in survival. These factors include differences in nutritional status, genetics, source hatchery husbandry, stage of smoltification, rates of latent pathogen infections, or previous exposure to contaminants and conditions of urbanized waterways.

Concentrations of LPAHs, HPAHs, and PCBs in juvenile chinook stomach contents were higher in LDW- and Puyallup estuary-collected fish than in fish collected from the Nisqually estuary or the Kalama Creek hatchery, suggesting elevated exposure. In 1989 and 1990, PCBs measured in livers and FACs were significantly higher in LDW fish than in fish from the Nisqually estuary or pooled data from all hatcheries. Butyltins were present at detectable concentrations (near detection limit) in LDW-collected chinook, but not in any other fish sampled in 1989 (the only year tested). Also, LDW-collected fish had significantly higher-level aryl hydrocarbon hydroxylase (AHH) activity and DNA adducts than fish from the Nisqually estuary or combined hatcheries (pooled data) for both years.

The authors suggested that the relatively higher contaminant burdens may be the cause for decreased survival in the laboratory of the LDW-collected fish relative to the Green River hatchery fish. However, additional study would be needed to verify these results due to the high mortality observed in all treatments and the potential influence of the factors cited above.

A.4.3.1.2 Survival following immunological challenge

This section discusses the only study available regarding survival of LDW-collected fish with subsequent immunological challenge (Arkoosh et al. 1998b). This study investigated potential effects of exposure to a model pathogen (*Listonella anguillarum*) on survival of juvenile chinook salmon collected from the LDW. Arkoosh et al. (1998b) collected juvenile chinook salmon from the LDW and Nisqually River, and from their respective upstream hatcheries (i.e., Green River and Kalama Creek, respectively), in the spring of 1993 and 1994. Analyses of bile, liver, and stomach contents showed that LDW salmon were generally exposed to higher concentrations of PAHs, PCBs, and DDTs than Nisqually or hatchery salmon. However, the only statistically significant difference in liver concentrations were HPAHs in 1993, and LPAHs in 1994 (Table A-4-18). PCBs, DDTs, and FACs in stomach contents were significantly higher in the LDW both years, with the exception of total PCBs in 1994 (Table A-4-19).

Table A-4-18. Contaminant concentrations in livers of juvenile chinook salmon from the LDW

Year/Sites	Number of Composites	Total LPAHs[a] (µg/g ww)	Total HPAHs[a] (µg/g ww)	Total PCBs[a] (µg/g ww)	Total DDTs[a] (µg/g ww)
1993					
Green River Hatchery	1	0.06z	0.022z	0.11zy	0.039z
Duwamish Waterway	3	2.9±3.0z	6.2±3.8y	0.47±0.23z	0.046±0.018z
Kalama Creek Hatchery	2	0.038±0.017z	0.021±0.01z	0.087±0.064y	0.019±0.005z
Nisqually Estuary	3	0.016±0.006z	0.010±0.004z	0.11±0.048zy	0.037±0.02z
1994					
Green River Hatchery	3	0.080±0.005z	0.026±0.001z	0.086±0.010z	0.024±0.008z
Duwamish Waterway	4	2.1±0.90y	2.8±1.70z	0.35±0.27z	.062±.084z
Kalama Creek Hatchery	na	na	na	na	na
Nisqually Estuary	1	0.0092z	0.0023z	0.025z	0.004z

SOURCE: Arkoosh et al. (1998b)

Note: For each year and within a column, values not sharing a common lowercase letter are significantly different.

na – data not available

[a] Values expressed as mean ± standard deviation

Table A-4-19. Contaminant concentrations in stomach contents of juvenile chinook salmon from the LDW

Year/Sites	Number of composites	Total PCBs[a] (µg/g ww)	Total DDTs[a] (µg/g ww)	Biliary FAC[a, b] (ng/BaP/mL bile)
1993				
Green River Hatchery	3	0.058±0.001z	0.019±0.002zy	1,110±23z
Duwamish Waterway	3	0.27±0.057y	0.042±0.011x	2,400±1,100y
Kalama Creek Hatchery	3	0.037±0.002z	0.012±0.00003z	710±230z
Nisqually Estuary	3	0.085±0.015z	0.024±0.003y	23±64z
1994				
Green River Hatchery	3	0.036±0.005z	0.015±0.001z	2,400±310y
Duwamish Waterway	4	0.090±0.047z	0.059±0.011y	4,300±1,100x
Kalama Creek Hatchery	3	27±1z	0.010±0.001z	1,000±19z
Nisqually Estuary	3	0.059±0.019z	0.0240.014	55±110z

SOURCE: Arkoosh et al. 1998b

Note: For each year and within a column, values not sharing a common lowercase letter are significantly different.

FAC – fluorescent aromatic hydrocarbon

[a] Values expressed as mean ± standard deviation

[b] Values expressed as ng BaP equivalents/mL bile

In 1993, field-collected juvenile salmon were challenged with three serial doses of *L. anguillarum*, and cumulative mortality after 7 days was compared within and between river systems (experiment 1). The study was repeated after fish were held in the laboratory for a 3-month period (experiment 2) to assess the duration of the effect of exposure on survival after fish were removed from potential chemical source. Experiment 1 was replicated in 1994 using two serial doses of *L. anguillarum* (experiment 3). Results are summarized in Tables A-4-20 and A-4-21.

For experiment 1, Arkoosh et al. (1998b) reported that LDW-collected salmon exhibited a significantly higher cumulative mortality at both four and seven days after exposure to the pathogen (at all pathogen concentrations) than salmon from the hatcheries or the Nisqually estuary. In experiment 2, no significant difference in susceptibility to *L. anguillarum* was observed between the two estuaries, except at the lowest *L. anguillarum* dose, four days after exposure, where the effect on LDW fish was higher. When experiment 1 was repeated the following year (experiment 3), salmon from the LDW exhibited a significantly higher cumulative mortality than Green River Hatchery fish four days after exposure to the lowest pathogen dose, but not after exposure to the higher dose. Seven days after exposure, no significant difference in survival was reported between the LDW-collected fish and the hatchery fish. Effects on reference Nisqually estuary fish were not significantly different from Kalama Creek Hatchery fish, but the Kalama Creek Hatchery fish also experienced high mortality.

Table A-4-20. Percent cumulative mortality of juvenile chinook salmon exposed to *L. anguillarum* at day 4

Expt. #	Dilution *L. anguillarum*	Percent Mortality							
		Green River Hatchery (GRH)	LDW	Kalama Creek Hatchery (KCH)	Nisqually Estuary (NE)	non-*L. anguillarum* Control -GRH	non-*L. anguillarum* Control - LDW	non-*L. anguillarum* Control - KCH	non-*L. anguillarum* Control - NE
1	$6.5X10^{-5}$	1	45[a,b]	5	3		18	0	0
	$1.8X10^{-4}$	2	60[a,b]	25	5	11			
	$4X10^{-4}$	7	63[a,b]	40	20				
2	$6.5X10^{-5}$	15	36[a,b]	40[c]	70		3	33	5
	$1.8X10^{-4}$	10	53[a]	57	55	13			
	$4X10^{-4}$	20	75[a]	[d]					
3	$2X10^{-5}$	9	32[a,b]	3	8	18	2	0	15
	$6X10^{-5}$	33	33	10	0				

Source: Arkoosh et al. (1998b)

[a] Significantly different from fish from corresponding hatchery ($p \leq 0.05$)

[b] Significantly different from fish from Nisqually estuary ($p \leq 0.05$)

[c] Salmon from the Kalama Creek Hatchery were not examined in duplicate. Fish from only one tank were exposed to this concentration of bacteria and analyzed.

[d] Salmon from the Kalama Creek Hatchery were not available for exposure at this concentration of bacteria.

Table A-4-21. Percent cumulative mortality of juvenile chinook salmon exposed to *L. anguillarum* at day 7

Expt. #	Dilution *L. anguillarum*	Percent Cumulative Mortality							
		Green River Hatchery (GRH)	LDW	Kalama Creek Hatchery (KCH)	Nisqually Estuary (NE)	non-*L. anguillarum* Control GRH	non-*L. anguillarum* Control LDW	non-*L. anguillarum* Control KCH	non-*L. anguillarum* Control NE
1	$6.5X10^{-5}$	1	66[a,b]	43	35	11	20	0	6
	$1.8X10^{-4}$	2	88[a,b]	78	34				
	$4X10^{-4}$	7	73[a,b]	75	46				
2	$6.5X10^{-5}$	35	62[a]	60[c]	65	26	18	33	15
	$1.8X10^{-4}$	30	58[a]	64	50				
	$4X10^{-4}$	48	75[a]	[d]					
3	$2X10^{-5}$	63	72[b]	52	35	10	5	3	15
	$6X10^{-5}$	75	80[b]	77	25				

Source: Arkoosh et al. (1998b)

[a] Significantly different from fish from the corresponding hatchery ($p \leq 0.05$)

[b] Significantly different from fish from the Nisqually estuary ($p \leq 0.05$)

[c] Salmon from the Kalama Creek Hatchery were not examined in duplicate. Only one tank of these fish exposed to this concentration of bacteria were analyzed.

[d] Salmon from the Kalama Creek Hatchery were not available for exposure at this concentration of bacteria.

Several issues, as described below, complicate the interpretation of these studies. Specifically:

- There was generally a high level of unexplained mortality in unchallenged control fish, averaging 12 to 15% with a maximum of 33% (Tables A-4-20 and A-4-21).
- Mortality for a given treatment was calculated by subtracting unchallenged control mortality from total mortality (based on duplicate control tanks of 20 fish per treatment in each experiment).
- *L. anguillarum* was verified as cause of death in up to 24% (13 of 55) of control mortalities (i.e., fish never exposed to *L. anguillarum*) (note: the percent attributable to *L. anguillarum* was not fully confirmed; see below).
- In all experiments, only a subsample of mortalities (approximately 1/3) were necropsied for the presence of *L. anguillarum*. Of the *L. anguillarum* exposed fish in experiments 1, 2 and 3 respectively up to 83%, 27%, and 66% of necropsied mortalities were not attributable to this pathogen. Because not all mortalities were tested, the actual mortality due to *L. anguillarum* cannot be accurately determined.
- Calculation of significant differences in survival at day 4 and 7 with this challenge model is not standard practice. *L. anguillarum* challenges should be run and analyzed only after a minimum of 14 days; typically, *L. anguillarum* mortality does not begin until day 4 or 5 of a challenge (Palm et al. 1998). Significant mortality in the first few days is usually due to unanticipated causes and selection of early challenge endpoints can substantially bias the results (Palm 1996).
- LDW fish were acclimated in the laboratory for approximately two weeks compared to approximately one month for Nisqually estuary fish and hatchery fish. In 1993, hatchery and Nisqually estuary fish were acclimated from 20 to 33 days longer than LDW fish. In 1994 hatchery and Nisqually estuary fish were acclimated from 13 to 22 days longer than LDW fish. Also, the LDW-collected fish were smaller at the start of the experiment (25% smaller by weight) than Green River Hatchery fish and 56% smaller by weight than Nisqually River estuary fish. Differences in size and/or age can mean that some groups are at different stages of smoltification or nutritional status. Group differences in either acclimation time or stage of smoltification can significantly affect challenge mortality levels.

In all three experiments, the high levels of uncontrolled and unexplained mortality, nonstandard challenge data analysis, including early challenge endpoints and subtraction of control mortality (Palm 1996; Amend 1981), lead to inconclusive results.

Arkoosh et al. (1998b) suggested immunosuppression in juvenile chinook salmon from the LDW may lead to increased susceptibility to infection by a virulent marine bacterium. The authors attributed the differences in susceptibility (as discussed above) to chemical exposure, based on generally higher chemical concentrations in LDW-captured fish. The variance in the stomach content data was much greater for LDW fish than for other treatment groups. Liver concentrations of DDTs were significantly elevated in LDW fish in 1993, but not in 1994, and liver PCB concentrations were not significantly different in either year.

In summary, although LDW-collected fish appear to have had higher exposure to sediment-associated chemicals, the immunological results of the study are inconclusive based on the following: 1) the high mortality observed in the unchallenged control fish, 2) verification of *L. anguillarum* as the cause of death in only a fraction of the mortalities from the challenged groups, and 3) the nonstandard challenge data analysis practices. Determination of any causative agents would require additional controlled exposure studies bracketing known environmental exposure concentrations delivered in the diet or water.

A.4.3.1.3 Growth

Results of LDW-specific investigations of potential effects on growth for juvenile chinook salmon were reported in Varanasi et al. (1993), Casillas et al. (1995a,b), and Casillas et al. (1998b).

Varanasi et al. (1993) collected juvenile chinook salmon from Puget Sound urban estuaries and hatcheries upstream of each estuary, held the fish in the laboratory, and compared survival (discussed above) and growth within and between river systems. Juvenile chinook salmon were sampled from hatcheries and the respective estuaries of the Green, Puyallup, and Nisqually Rivers in 1990. In 1991, fish were sampled only from the Green River hatchery and the LDW. Whole fish and liver tissues, stomach contents, blood and bile were sampled for organic chemical and biochemical analyses.

The experiment was designed to compare growth (within and between river systems) after fish were held in the laboratory for 120 days. After early termination of the experiment at 40 days in 1990 (because of bloating in fish from all treatments), a significantly smaller increase was observed in length (7 mm vs. 9 mm increase), but not in weight, for LDW estuary fish when compared to Green River hatchery fish. At the same time, a significantly smaller increase in both length and weight was observed in fish from the Nisqually estuary when compared to Kalama hatchery fish (the control river system). Effects on Nisqually estuary fish were attributed to a kidney fluke. Note, the LDW fish were 19% smaller in weight and 14% smaller in length than Green River hatchery fish at the start of the experiment. Therefore, their nutritional status (vitamin stores etc.) and potential carrier states of disease may have influenced the analysis.

In 1991, no fish were tested from the Nisqually reference area and the experiment was terminated early at 84 days because of increasing mortality in the controls. A significantly smaller increase in length but not weight was reported for fish from the LDW when compared to the Green River Hatchery fish, but no reference area results were available for comparison. Thus, although this study suggests LDW fish may experience increased chemical exposure relative to hatchery and control fish,[84] the results reported for growth would have to be verified due to high mortality in all treatments both years, the differing initial sizes of the fish, and, as the authors acknowledge, lack of an appropriate reference estuary for the 1991 study. Wild fish may be more stressed or not feed as well under laboratory conditions using artificial diets compared to fish reared for generations (a portion of their life) in hatchery systems.

Casillas et al. (1995a,b) reported that a growth experiment similar to that of Varanasi et al. (1993) was carried out in 1993, comparing fish from the LDW and Green River hatchery to those of the Skokomish River estuary and hatchery. Casillas et al. (1995a,b), however, only reported the conclusions from the studies. The authors stated there were additional changes to husbandry practices to increase survival. The authors reported that in 1993, they did not experience the problems with fish husbandry experienced in 1990-91. They reported fish from the LDW had significantly poorer growth than fish from the Green River hatchery, whereas growth of fish from the Skokomish River estuary did not differ from that of Skokomish hatchery fish. Data from these studies have not been published in any available report; thus, results from this experiment cannot be verified or used for the ERA unless data are made available for review.

A.4.3.2 English sole

Several studies with English sole have been conducted in the LDW and the greater Puget Sound area (Table A-4-22). These studies involved either direct field exposure, laboratory exposure to field-collected sediments, or IP injection with organic sediment extracts. As discussed, because of the uncertainties associated with exposure to chemical mixtures, these studies cannot be used to derive NOECs or LOECs for the risk characterization (Section A.7.2.1).

Regional studies conducted in the Hylebos Waterway (Malins et al. 1984, 1985a) and Eagle Harbor (Malins et al. 1985b; Kubin 1997; Schiewe et al. 1991) are acknowledged in this section because they involved English sole and may contribute to the weight-of-evidence analysis. However, these studies are not discussed in detail because fish were exposed to chemical mixtures that are not specific to the LDW. In the case of IP injection studies, the exposure route is not ecologically relevant.

---

[84] Stomach contents of fish from the LDW had significantly higher concentrations of PAHs and PCBs (but not other chlorinated hydrocarbons [hexachlorobenzene, lindane, heptachlor, aldrin, alpha-chlordane, dieldrin, DDE, DDD, and DDT]) than fish from the Green River Hatchery or the Nisqually River estuary.

Table A-4-22. LDW-specific and regional English sole growth, reproductive effects, and survival studies

Study	Study Type	Exposure	Effects Endpoints
LDW-specific studies			
Johnson et al. 1988,1997,1999	field	field	reproductive effects
Casillas et al. 1991	field	field	reproductive effects
Rhodes et al. 1987	field	field	mortality - lesions
Johnson and Landahl 1994	field	field	mortality - lesions
Regional studies			
Kubin 1997	lab	sediment	growth
Malins et al. 1984, 1985a, 1985b	field	field	mortality – lesions
Schiewe et al. 1991	lab	IP injection	mortality – lesions

In this section, studies conducted with English sole collected from the LDW and other nearby Puget Sound locations are discussed. This discussion is organized according to survival, growth, and reproduction endpoints. Studies involving liver lesions and English sole are considered under the survival endpoint.

A.4.3.2.1 Survival

No studies have been conducted to directly examine survival of LDW-collected English sole relative to those collected from reference areas. However, several studies were available regarding lesion prevalence. These studies are discussed below. Johnson and Landahl (1994) examined the relationship between lesion prevalence and population-level effects in a study of estimated annual mortality rates from heavily and minimally contaminated sites throughout Puget Sound, including the LDW. They concluded that sole mortality rates from contaminated sites with higher liver lesion prevalence were not significantly greater than mortality rates for sole from Puget Sound as a whole. Johnson and Landahl (1994) also examined English sole population structure. Maximum age and percent of older fish (15 years or older) in the population were determined for English sole with and without lesions, as well as different embayment populations. There was no evidence of increased age-related mortality in fish with lesions or in populations associated with areas with higher levels of chemicals. In fact, the maximum age class of sole and percent older fish in the population tended to correlate with elevated concentrations of PAHs and PCBs in sediment. The authors concluded that mortality associated with exposure to sediment chemicals appeared to be insignificant compared to other factors that effect English sole populations, such as fishing pressure, predation, and fluctuations in food supply. The results of this study provided no evidence that exposure of English sole to sediment-associated chemicals in urban areas of Puget Sound has any adverse effect on survival of the English sole population.

Rhodes et al. (1987) investigated the relationships between the prevalence of lesions and age, gender, and site season, and year of capture in English sole collected from

8 sites, including the LDW over 5 years (1979-1984 with no sampling in 1981). Fish from the LDW were more likely to have lesions than the reference fish population (Port Madison, WA). The location of capture was reported to be one of the most important factors in predicting lesion occurrence. Age of the fish and season of capture were also important variables in predicting lesion occurrence.

Malins et al. (1984), as part of a multi-year study, investigated the relationships between sediment contaminant concentrations and diseases in bottom-dwelling fish from Puget Sound. Hepatic neoplasms were found to be mainly confined to fish in urban areas (LDW, Commencement Bay (Hylebos Waterway), and the harbor area of Everett, WA).

A.4.3.2.2 Growth

The growth endpoint has not been assessed with English sole collected from the LDW. Although laboratory studies with indirect and direct exposure to Eagle Harbor sediments suggest chemical exposure may depress English sole growth (Rice et al. 2000; Kubin 1997), there is no evidence of suppressed growth in subadult sole collected from Eagle Harbor, a site with elevated sediment PAH concentrations. Juvenile sole collected from Eagle Harbor were significantly larger than sole of the same age collected from an adjacent site outside Eagle Harbor (Johnson et al. 1998). However, as in any field study, determining cause and effect is difficult.

A.4.3.2.3 Reproduction

Potential reproductive effects on English sole exposed to sediment-associated chemicals in the LDW and other sites throughout Puget Sound have been assessed using a variety of endpoints. Concentrations of the female reproductive hormone 17 beta-estradiol in plasma have been analyzed (Johnson et al. 1988, 1997, 1999) and ovarian development (Johnson et al. 1988, 1993), spawning success (Casillas et al. 1991), and fecundity (Johnson et al. 1997) have been assessed.

English sole from the LDW have been cited as having inhibited gonadal development (Johnson et al. 1988), depressed plasma estradiol and reduced ovarian production *in vitro* (Johnson et al. 1988, 1993), and reduced spawning success (Casillas et al. 1991). The authors suggested these effects occur as a result of elevated concentrations of aromatic and chlorinated hydrocarbons present in LDW and Eagle Harbor sediments. These elevated concentrations have also been suggested as significant risk factors for development of these reproductive abnormalities (Johnson et al. 1988; Casillas et al. 1991).

Johnson et al. (1988, 1997, 1999) reported that female English sole from urban estuaries are less likely to enter vitellogenesis and have lower plasma concentrations of the female reproductive hormone 17 beta-estradiol than do sole from areas with lower levels of chemical exposure. According to Johnson et al. (2002), at sites with sediment total PAH concentrations less than 0.5 mg/kg dw, approximately 80 to 90% of adult

females undergo normal gonadal development, while in the LDW, the Hylebos Waterway, and Eagle Harbor, the percentage declines to 40 to 60%.

Johnson et al. (1988) examined reproductive effects at four sites: Eagle Harbor, Sinclair Inlet, Port Susan, and the LDW. English sole were collected over two winters from November through January, the period in which vitellogenesis occurs, but before substantial migration has taken place. Larger, presumed sexually mature female fish (300 mm or greater) were collected, and subjected to biomarker and chemical exposure assessment, as well as ovarian pathology. Elevated hepatic lesions, bile FACs, and liver PCB concentrations were measured in LDW fish relative to concentrations in reference fish from Port Susan and Sinclair Inlet. The LDW fish also exhibited a greater degree of inhibited ovarian development relative to the reference fish in 1986, but not in 1987. Plasma estradiol levels measured in the LDW fish were significantly lower than those measured in reference fish in 1987, but not in 1986. There were no significant differences in vitellogenin level or gonadosomatic index measured in the LDW and Eagle Harbor fish compared to reference fish (Port Susan and Sinclair Inlet) either year.

Johnson et al. (1988) found the best predictor of potential reproductive effects was hepatic AHH activity and the overall condition factor. These indicators combined accounted for 34 percent of the observed variability in the occurrence of ovarian development. The authors concluded that the remainder of the observed variability (66%) was attributable to factors not measured in this study, such as unmeasured chemicals, genetic variation, health, or seasonal variation in the spawning cycle (Johnson et al. 1988). In conclusion, measures of reproductive effects presented in this study were highly variable with no clear indication that observed effects could be attributed to exposure to sediment chemicals.

Casillas et al. (1991) collected gravid English sole from the same four Puget Sound sites (Port Susan, Sinclair Inlet, the LDW, and Eagle Harbor) investigated by Johnson et al. (1988). These field-collected fish were artificially induced to spawn in the laboratory; spawning success was significantly lower in fish from Eagle Harbor and the LDW relative to the reference fish (Casillas et al. 1991). A model was developed to relate plasma estradiol and vitellogenin concentrations, fish length, site of capture, condition factor, presence of liver lesions, and time of capture to probability of spawning. The only variables found to have a significant effect on the probability of spawning were initial plasma estradiol and vitellogenin concentrations, time of capture, and capture at Eagle Harbor. Exposure in the LDW was not found to be a significant factor in the model. The spawning success was quite sensitive to time of capture; fish collected in late January were more likely to spawn than those collected in early January.

In addition, Johnson et al. (1997) performed a study on age- and size-specific patterns of egg production in Puget Sound English sole by collecting vitellogenic female English sole from the same four sites (Eagle Harbor, Sinclair Inlet, Port Susan, and the

LDW). These fish were not actively spawning, so fecundity measurements estimate potential rather than actual fecundity. Differences in potential fecundity were compared to differences in collection site, chemical contamination, exposure, and nutritional status. Fecundity was assessed as egg weight from preserved ovaries. Fish length was reported to be the strongest indicator of fecundity. LDW fish had significantly higher fecundity than Port Susan fish, and LDW fish had a higher age-specific fecundity as well. The LDW fish also produced larger numbers, but smaller eggs, relative to the reference fish.

In conclusion, there is some evidence that English sole collected from the LDW may exhibit reproductive differences from English sole collected from reference sites. These results are included in the risk characterization discussion (Section A.7.3). Johnson et al. (2002), in which reproductive impacts in English sole from PAH concentrations in field sediments were assessed using a hockey stick statistical approach, will be discussed in the Phase 2 ERA.

A.4.4 Summary of Fish Assessment

A.4.4.1 Summary of fish exposure assessment

In Section A.4.1, exposure of fish to sediment-associated COPCs was assessed using two approaches depending on the potential bioaccumulation of the COPC:

1) PCB, DDT, TBT, or mercury concentrations in ROC tissue (critical tissue residue approach)
2) Estimated and measured copper and arsenic concentrations in benthic invertebrate prey (dietary approach)

Limited data were available for each approach, and assumptions were required regarding substitution of tissue data from one fish for another, use of fillet data, and the ability of measured concentrations of arsenic and copper in amphipods collected near Kellogg Island to approximate spatially weighted average dietary exposure by fish. Table A-4-23 provides a summary of exposure concentrations for each ROC/COPC pair.

Table A-4-23. Exposure concentrations for ROC/COPC pairs identified in Table A-4-1

ROC	Dietary Concentration (mg/kg, dw)			Whole Body Residue (µg/g, ww)			
	Arsenic	Copper	PAHs	Mercury	TBT	DDTs	PCBs
Juvenile chinook	8.2	166	165 [a]	0.088	0.18	0.041	0.17
Bull trout	5.8	9.1	ne	0.44	0.18	0.14	8.1
English sole	32	121	3.1	0.076	0.019	0.0069	2.3

ne – Not evaluated in the EEA (screened out in the problem formulation)

[a] Dry weight calculated assuming 80% moisture content in stomach contents.

A.4.4.2 Summary of fish effects assessment

The effects assessment was divided into two sections. Section A.4.2 presented an overview of the available literature involving laboratory studies with controlled exposures to single COPCs. Ranges of NOECs and LOECs from these studies were reviewed and specific NOECs and LOECs were selected based on taxonomic similarity of test species to ROCs, as well as the relevance of the study exposure conditions. A summary of selected NOECs and LOECs is presented in Tables A-4-24 through A-4-26.

Table A-4-24. TRVs for fish ROC/COPC pairs (survival endpoint)

Dietary TRVs (mg/kg dw)						
	Arsenic		Copper		PAHs	
ROC	NOEC	LOEC	NOEC	LOEC	NOEC	LOEC
Juvenile chinook	na	na	730	na	1,000 (ww)	na
Bull trout	na	na	730	na	ne	ne
English sole	na	na	730	na	1,000 (ww)	na

Critical Body Residues (µg/g ww)								
	Mercury		TBT		DDTs		PCBs	
ROC	NOEC	LOEC	NOEC	LOEC	NOEC	LOEC	NOEC	LOEC
Juvenile chinook	29	na	0.17	1.7	1.8	3.0	120 [a]	645
Bull trout	0.2	0.47	0.17	1.7	1.8	3.0	27	46
English sole	0.2	0.47	0.17	1.7	0.62	3.65	27	46

NE – Not evaluated in the EEA (screened out in the problem formulation)

na – No TRV available because of lack of relevant toxicity data

[a] *Value represents NOEC for both survival and survival following immunological challenge*

Table A-4-25. TRVs for fish ROC/COPC pairs (growth endpoint)

Dietary TRVs (mg/kg, dw)						
	Arsenic		Copper		PAHs	
ROC	NOEC	LOEC	NOEC	LOEC	NOEC	LOEC
Juvenile chinook	20	30	684	700	100	1,000
Bull trout	20	30	684	700	ne	ne
English sole	20	30	8	16	47	116

Critical Body Residues (µg/g, ww)								
	Mercury		TBT		DDTs		PCBs	
ROC	NOEC	LOEC	NOEC	LOEC	NOEC	LOEC	NOEC	LOEC
Juvenile chinook	8.63	10	na	na	7.6	na	70	120
Bull trout	0.8	1.31	na	na	7.6	na	0.98	3.7
English sole	0.8	1.31	na	na	7.6	na	0.98	3.7

ne – Not evaluated in the EEA (screened out in the problem formulation)

na – No TRV available because of lack of relevant toxicity data

Table A-4-26. TRVs for fish ROC/COPC pairs (reproduction endpoint)

Dietary TRVs (mg/kg, dw)						
	Arsenic		Copper		PAHs	
ROC	NOEC	LOEC	NOEC	LOEC	NOEC	LOEC
Juvenile chinook	ne	ne	ne	ne	ne	ne
Bull trout	na	na	na	na	na	na
English sole	na	na	na	na	na	na

Critical Body Residues (µg/g, ww)								
	Mercury		TBT		DDTs		PCBs	
ROC	NOEC	LOEC	NOEC	LOEC	NOEC	LOEC	NOEC	LOEC
Juvenile chinook	ne	ne	ne	ne	ne	ne	ne	ne
Bull trout	0.21	2.1	0.18	1.8	0.3	3.0	1.9	9.3
English sole	0.21	2.1	0.18	1.8	0.3	3.0	1.9	9.3

ne – Not evaluated in the EEA (screened out in the problem formulation)

na – No TRV available because of lack of relevant toxicity data

Section A.4.3 summarized regional studies in which juvenile chinook salmon or English sole were exposed to a mixture of chemicals either through field exposure or through IP injection of sediment extracts. No NOECs or LOECs were selected from these studies.

A.5 Exposure and Effects Assessment: Wildlife

This section presents the exposure and effects assessment for wildlife receptors. In the problem formulation, five wildlife species were selected as ROCs in the LDW: spotted sandpiper, great blue heron, bald eagle, river otter, and harbor seal. COPCs for these ROCs were selected based primarily on the results of the wildlife risk assessment in King County (1999c), and additional screens conducted in the problem formulation. The COPCs for each specific wildlife receptor are presented in Table A-5-1. This section presents a detailed evaluation of potential exposure through a basic food web model, and reviews effects levels identified in the scientific literature associated with chemical exposures. In addition, PCB concentrations measured in great blue heron eggs collected from the West Seattle colony in 1998 are presented along with effects data from the scientific literature.

Table A-5-1. ROC/COPC pairs to be evaluated for wildlife

ROC	PCBs	BEHP	As	Cu	Pb	Hg	Zn
Sandpiper	x	x		x	x		x
Heron	x				x	x	
Eagle	x				x	x	
Otter	x		x		x		
Seal	x						

A.5.1 Exposure Assessment

This exposure assessment presents the methods used and results of calculated potential dietary doses of COPCs to ROCs in the LDW. In the risk characterization (Section A.7.3.1), these exposure doses are compared to doses at which effects have been observed in laboratory studies. The effects concentrations from laboratory studies are discussed in Section A.5.2.

A.5.1.1 Approach

In this assessment, estimates of daily chemical exposures for each receptor are calculated for two exposure pathways: ingestion of food and incidental ingestion of sediment. Other pathways considered in the conceptual site model in the problem formulation were determined to be insignificant.[85] The exposure dose estimates were calculated using the following equation:

[85] Direct (or dermal) contact with sediment was considered a complete exposure pathway, although risks from sediment contact are considered to be insignificant relative to those from ingestion (EPA 2000a). Direct contact with water was also considered a complete exposure pathway, but also was assumed to be insignificant because feathers on birds and fur on mammals limit direct contact of skin with contaminated media. Water ingestion, although a complete and quantifiable pathway, was not included in the exposure estimate because the King County Wildlife Risk Assessment found that less than 0.5% of the calculated risk for each ROC/COPC pair in the LDW was contributed by the water ingestion pathway (King County 1999c).

$$\text{Exposure Dose} = \frac{[(DFC \times C_{food}) + (DSC \times C_{sed})] \times SUF}{BW} \qquad \textit{Equation 5-1}$$

Where:

Exposure Dose	=	COPCs ingested per day via food and sediment (mg COPC/kg body weight/day)
DFC	=	daily food consumption rate (kg food day dw)
C_{food}	=	concentration in prey items (mg COPC/kg food dw)
DSC	=	daily sediment consumption rate (kg sediment/day dw)
C_{sed}	=	concentration in sediment (mg COPC/kg dw)
SUF	=	site use factor (unitless)
BW	=	wildlife species body weight (kg ww)

The site use factor is the fraction of time that a receptor spends foraging in the LDW compared to other areas. Site use factors, daily food consumption rates, and body weights vary among ROCs and are described in Section A.5.1.3. The daily food consumption rates and body weights were obtained from the scientific literature for each receptor. All COPCs were assumed to be 100% bioavailable. The daily sediment consumption rate was calculated by multiplying the DFC by the proportional sediment ingestion rate for the ROC. The sediment ingestion rate was largely based on best professional judgment (see Section A.5.1.3).

The chemical concentration in food was calculated from concentrations in each component of the wildlife species diet and each component's fraction of the diet. For example, the concentration in food for an ROC that might ingest fish and amphipods would be estimated as follows:

$$C_{food} = (C_f \times F_f) + (C_a \times F_a) \qquad \textit{Equation 5-2}$$

Where:

C_{food}	=	concentration in prey items (mg COPC/kg food dw)
C_f	=	concentration in fish tissue (mg COPC/kg tissue dw)
C_a	=	concentration in amphipod tissue (mg COPC/kg tissue dw)
F_f	=	fraction of the wildlife species diet that is fish (kg fish/kg food)
F_a	=	fraction of the wildlife species diet that is amphipods (kg amphipods/kg food)

The dietary fraction of each prey item was based on information from the scientific literature. Available site-specific data for prey species were used to estimate COPC concentrations in prey items in Section A.5.1.2.

A.5.1.2 Prey tissue, sediment, and egg data

A.5.1.2.1 Prey tissue

Tissue data were available for six potential prey species of ROCs in the LDW. These data are described in Table A-5-2, along with the location, number of samples, and

tissue type analyzed. A map showing the locations of tissue collected is presented in Map A-2-1 (Attachment A.1). All samples were composite samples.

Table A-5-2. Tissue samples used in estimating exposure to wildlife

Tissue Type	Location	Number of Samples	Number of Organisms per Composite	Tissue Analyzed	Collected By
Amphipods	Kellogg Island W. Marginal Way	2 2	~2,000	whole body	King County
Dungeness crab	LDW Transect	2 1	3 3	edible meat hepatopancreas	King County
Mussels[a]	Brandon St. Duwamish/Diagonal Kellogg Island Slip 4 Terminal 107	6 8 3 3 2	50	soft parts	King County
Shiner surfperch	LDW Transect	3	10	whole body	King County
English sole	LDW Transect	3	20	whole body	King County
Juvenile chinook salmon	Kellogg Island[b] Slip 4[c]	23 3	2–10 5–7	whole body	NMFS

na – Not available: number of organisms per composite was not clear in report

[a] Wild mussels, not transplants.

[b] 14 composite samples from Varanasi et al. (1993); 7 composite samples from NMFS (2002); and 2 composite samples statistically constructed from individual samples to maximize the amount of usable data, as described in Section A.4.1.1.

[c] 2 composite samples from NMFS (2002) and 1 composite sample statistically constructed from individual samples to maximize the amount of usable data, as described in Section A.4.1.1.

Tissue concentrations of COPCs for each prey type relevant to a ROC are shown in Table A-5-3. This table presents concentrations for minimum, maximum, arithmetic mean, and a one-sided 95 percent upper confidence limit (UCL) on the mean. Given the small number of data points, it was difficult to determine the underlying distribution of the data. For the purposes of the Phase 1 exposure assessment, a normal distribution was assumed. Accordingly, the confidence limit on the mean was estimated using the t-distribution and the sample estimates for mean and variance:

$$95\text{th UCL} = \bar{x} + \left(t_{.05(1),n-1}\right)\left(\frac{s}{\sqrt{n}}\right) \qquad \textit{Equation 5-3}$$

Where:

x = mean concentration
$t_{.05(1),\,n-1}$ = the 95th percentile of the t-distribution with n-1 degrees of freedom
n = number of measurements
s = standard deviation

For samples with undetected values, one-half the detection limit was substituted to calculate the mean and the 95% UCL on the mean.

Table A-5-3. Concentrations of COPCs (mg/kg dw) in prey species[a]

Chemical	No. Composite Samples	No. Organisms per Composite	Minimum	Maximum	Mean	95% UCL
Amphipods						
PCBs	4	~2,000	0.594	2.28	1.26	**2.18**
Copper	4	~2,000	54.6	167	104	**166**
Lead	4	~2,000	5.3	41.5	20.8	**41.4**
Zinc	4	~2,000	43.9	147	75.9	**132**
BEHP	4	~2,000	0.067	2.97	1.01	**2.62**
Dungeness crab[b]						
PCBs	2	1–3	1.69	**1.79**	1.74	nc
Arsenic	2	1–3	33.7	**60.3**	47.0	nc
Lead	2	1–3	1.08	**1.20**	1.14	nc
Mercury	2	1–3	0.397	**0.539**	0.468	nc
Mussels						
PCBs	22	50–100	0.054	0.250	0.153[c]	**0.179**
Arsenic	22	50–100	1.42	4.46	3.48[c]	**3.78**
Lead	22	50–100	0.55	3.01	1.88[c]	**2.20**
Mercury	22	50–100	0.037	0.095	0.050[c]	**0.056**
Shiner surfperch						
PCBs	3	10	1.47	**2.57**	2.07	3.00
Arsenic	3	10	4.33	**5.79**	5.30	6.72
Lead	3	10	0.596	**0.729**	0.700	0.791
Mercury	3	10	0.294	**0.367**	0.325	0.369
English sole						
PCBs	3	20	3.22	**9.15**	6.06	11.1
Arsenic	3	20	22.4	**25.8**	23.8	26.8
Lead	3	20	0.53	**0.88**	0.80	1.08
Mercury	3	20	0.239	**0.338**	0.279	0.367
Juvenile chinook salmon						
PCBs	26[d]	2–10	0.071	1.08	0.429	**0.667**
Arsenic	na	na	na	na	na	na
Lead	na	na	na	na	na	na
Mercury	na	na	na	na	na	na

Values in **bold** were used in the exposure calculations

[a] Dry weights were calculated using measured moisture content with the exception of mussels, perch, and salmon, which were not analyzed for moisture content. For those concentrations, the average moisture content in English sole was used (76%).

[b] Only two samples were collected so the 95% UCL was not calculated. Concentrations are calculated using both edible meat and hepatopancreas tissues—meat was assumed to constitute 85% and hepatopancreas was assumed to constitute 15% of total edible tissue wet weight.

[c] Weighted average concentration: first the average concentration of each station was calculated, then the average of these numbers was used.

[d] Three of the composite samples were constructed from individual samples at Kellogg Island and Slip 4, as discussed in Section A.4.1.1.

na No data available

nc Not calculated

The 95% UCL provides a conservative estimate of average exposure concentrations in prey. However, in cases where the variability in the data is extremely high, which may occur with small data sets such as this one, the maximum value may be exceeded by the 95% UCL. In these cases, EPA (1989) recommends the maximum value be used. Therefore, this assessment used the 95% UCL, unless it was higher than the maximum, in which case the maximum concentration was used. The crab data set consisted of only two samples, so the maximum concentration was used. Concentrations in crab were calculated with both edible meat and hepatopancreas data using a weighted concentration (assuming 15% of the crab body weight is hepatopancreas).

A.5.1.2.2 Sediment

Sediment data used to estimate exposure of wildlife in the LDW included approximately 1,200 surface sediment samples, as described in Section A.2.4. Sediment concentrations were spatially averaged within two designated foraging areas; one area consisting of the entire LDW and the other consisting of intertidal areas only.[86] It was assumed that bird ROCs ingest sediment from intertidal areas only (Section A.5.1.3), whereas mammal ROCs ingest sediment from both intertidal and subtidal areas. Map A-2-2 shows designated intertidal areas of LDW. Spatial analysis of sediment data was conducted using the Thiessen polygon method as previously described in Section A. 3.1.1.2. The spatially weighted average (SWA) concentrations, presented in Table A-5-4, were the sediment concentrations used in the exposure calculations (Equations 5-1 and 5-2). The chemicals presented for the entire LDW are COPCs identified for all wildlife ROCs, while chemicals presented for the intertidal area are COPCs for bird ROCs (see Table A-5-1). The use of SWAs is justified by the large home range of all ROCs relative to the LDW, with the exception of spotted sandpiper, which likely have a smaller home range than the other wildlife ROCs (see Section A.5.1.3.1).

[86] Spatially weighted averages were not calculated for all intertidal areas because the low sampling density in some areas would provide higher relative weight to those concentrations that could result in disproportionate weighting of some measurements and potentially bias the average values. Therefore, only the areas with relatively high sampling density were included, as shown on Map A-2-2. The uncertainty of excluding these areas is discussed in Section A.7.3.2.2.

Table A-5-4. Spatially weighted average sediment concentrations (mg/kg dw) of COPCs in the LDW

CHEMICAL	SWA CONCENTRATION (mg/kg dw)
Entire LDW	
PCBs	0.37
Arsenic	12
Lead	48
Intertidal LDW	
PCBs	0.73
Copper	48
Lead	61
Mercury	0.12
Zinc	110
BEHP	0.27

A.5.1.2.3 Eggs

Five eggs were collected from the great blue heron colony in West Seattle by USFWS in 1998 and were analyzed for PCBs at the National Marine Fisheries Service (NMFS) laboratory using congener-specific analysis[87] (Krausmann 2002b). Two of the five samples (WSGBH 2 and WSGBH 5) contained well-developed embryos, and therefore, were divided into two subsamples (one containing the yolk sac and the other containing the embryo plus remaining yolk sac) and extracted individually. The yolk sac portion of sample WSGBH 5 was lost during the sample cleanup.

Total PCB concentrations in the eggs are presented in Table A-5-5. Individual weights of subsamples for WSGBH 2 and WSGBH 5 were not measured, so a PCB concentration in the whole egg could not be directly calculated. Instead, the whole egg PCB concentration was estimated using assumptions about the proportional weights of the yolk sac and the embryo plus remaining yolk sac. Although the developmental stage of the embryos was not measured, the concentration of PCBs in embryo relative to yolk in sample WSGBH 2 suggests a substantial movement of PCBs into the chick, based on a comparison to heron embryo:yolk PCB ratios measured in earlier regional studies (Norman 2002c). This relatively high embryo PCB concentration indicates a late developmental stage based on other studies that have measured concentrations of PCBs in heron embryo and yolk samples at various developmental stages (Norman 2000c). In embryos of this age from these other studies, 58 to 65% of the total egg weight was contained in the chick embryo portion of the egg and the remainder in the yolk sac (Norman 2002c). Therefore, to calculate whole egg PCB concentration in

[87] The analytical method used by NMFS was a high performance liquid chromatography method coupled with photodiode array detection (HPLC/PDA). This is a non-standard method that had not previously been used by NMFS for analyzing bird eggs for PCB congeners (Krausmann 2002b). Uncertainties associated with the analytical method are discussed in Section A 7.3.2.2.

sample WSGBH 2, it was assumed, based on Norman (2002c), that the embryo was 58% of the total weight and the yolk was 42%. The whole egg PCB concentration in sample WSGBH 5 could not be estimated because the yolk sac portion of this sample was lost.

Table A-5-5. Concentrations of total PCBs and TEQs in great blue heron eggs

Sample	Total PCBs (mg/kg ww)	TEQs Using TEFs from Ahlborg (1994) (µg/kg ww)	TEQs Using WHO TEFs (µg/kg ww)
Whole egg			
WSGBH 1-H	1.9	0.045	0.020
WSGBH 3-H	2.1	0.053	0.022
WSGBH 4-H	12	0.48	0.34
Embryo plus remaining yolk sac[a]			
WSGBH 2-H	30	1.1	0.77
WSGBH 5-H	4.6	0.1	0.044
Yolk sac[a]			
WSGBH 2-Y	70	2.7	2.0
Estimated whole egg			
WSGBH 2-H and 2-Y	47	1.8	1.3

[a] Two of the five samples (WSGBH 2 and WSGBH 5), contained well-developed embryos, so they were divided into two subsamples (one containing the yolk sac and the other containing the embryo plus remaining yolk sac) and extracted individually. The yolk sac for Sample WSGBH 5 was lost during the sample cleanup step.

In addition to total PCB egg concentrations presented in Table A-5-5, 2,3,7,8-tetrachlorodibenzo-*p*-dioxin (TCDD) equivalents (TEQs) were calculated from the PCB congener data. TEQs were calculated for each sample as the sum of the dioxin-like toxicity of the PCB congeners to yield a single concentration equivalent to the toxicity of a similar concentration of TCDD. The TEQ approach is based on results of numerous studies of laboratory animals and cell culture toxicity tests that demonstrate that some of the most toxic planar halogenated hydrocarbons cause similar adverse effects but have different potencies. In this approach, a comparison of the toxicities of key planar halogenated hydrocarbons with the toxicity of TCDD was used to develop a TEF for each compound. A total TEQ for a sample was calculated by summing TEFs for individual congeners, as follows:

$$TEQ = \sum(TEF_i[Congener_i]) \qquad \textit{Equation 5-4}$$

Where:

TEQ = Weighted sum of dioxin-like toxicity
TEF_i = TCDD equivalent factor for $congener_i$
Congener = Concentration of dioxin-like congener in tissue

A number of sets of TEFs are available; USFWS calculated TEQs using TEFs from Ahlborg et al. (1994) and the World Health Organization (WHO; Van den Berg et al. 1998). The Ahlborg et al. (1994) TEFs were based on mammalian data, whereas the

WHO TEFs were developed specifically for birds. Therefore, for risk estimation, TEQs calculated with WHO TEFS were used.[88] In the risk characterization, the highest concentrations of total PCBs and PCB-TEQs were compared to bird egg concentrations from the scientific literature associated with adverse effects to estimate risk. These highest measured concentrations were in the sample in which the whole egg concentration was estimated. Effects of the uncertainty of estimated concentrations on risk assessment are discussed in Section A.7.3.2.2. Effects concentrations from the literature are presented in Section A.5.2.2.

A.5.1.3 Exposure assumptions

This section presents values used in Equations 5-1 and 5-2 to calculate the daily exposure dose for each ROC, including body weight, food consumption rate, site use factor, and dietary composition. Table A-5-6 summarizes these values and the following sections provide details of exposure factor assumptions for each ROC.

Table A-5-6. Exposure factor values for each ROC

ROC	Sex	Body Weight (kg)	Food Consumption Rate (kg/day dw)	Site Use Factor (unitless)	Diet Composition	Sediment Consumption
Spotted sandpiper	M	0.0379	0.0060	1	100% amphipods	18% of food consumption rate (intertidal)
	F	0.0471	0.0074			
Great blue heron	F	2.20	0.0931	1	100% perch	2% of food consumption rate (intertidal)
Bald eagle	F	5.24	0.151	0.25 and 1	100% fish (equal amounts of perch, sole, salmon) [a,b]	1% of food consumption rate (intertidal)
River otter	F	7.9	0.264	1	88% fish (equal amounts of perch, sole, salmon)[a,b] 10% crab 2% mussel	2% of food consumption rate (all areas)
Harbor seal	F	76.5	0.577	0.33	100% fish (equal amounts of perch, sole, salmon) [b]	2% of food consumption rate (all areas)

[a] No lead or mercury data were available for salmon, so the highest concentrations from the perch or sole data were used (lead from English sole and mercury from perch).

[b] For salmon, juvenile chinook data were used. Although eagle, otter, and seal may eat adult salmon, PCB concentrations were higher in juvenile chinook whole-body samples than in adult chinook or coho fillets, so juvenile data were used. Further, adult salmon tissue concentrations may not reflect exposure in the LDW, as discussed in the problem formulation. Lead, mercury and arsenic, the other COPCs for eagle, otter, or seal, were not measured in adult salmon fillets.

[88] It should be noted that WHO TEFs were not available for congeners 170 and 180, so mammalian TEFs were used by Krausmann (2002b) to calculate TEQs for these congeners.

A.5.1.3.1 Spotted sandpiper

Body Weight and Daily Food Consumption

Representative body weights for adult male and female spotted sandpipers (Table A-5-6) were obtained from a study by Maxson and Oring (1980), as cited in EPA (1993b). The daily food consumption rate was estimated as a function of the metabolic rate and the caloric content of the prey using the following equation:

$$DFC = \frac{FMR}{ME} \times \frac{0.001\,kg\,food}{g\,food} \qquad \textit{Equation 5-6}$$

Where:

DFC = daily food consumption rate (kg food/day dw)
FMR = free-living metabolic rate (kcal/day)
ME = average metabolizable energy of the total diet (kcal/g food dw)

The free-living metabolic rate (FMR) of the common sandpiper (2.83 kJ/g bw/day or 0.676 kcal/g bw/day) from Nagy et al. (1999) was used to derive male and female FMRs for spotted sandpiper (25.6 and 31.8 kcal/day, respectively). The ME value (4.3 kcal/g dw) was used for insects ingested by birds from Nagy (1987). The final calculated male and female food consumption rates are 0.0060 and 0.0074 kg/day dw, respectively (Table A-5-6).

Composition of Diet

Spotted sandpipers feed along the sandy or muddy edges of inlets, creeks, and ponds. Their diet is composed primarily of terrestrial and marine invertebrates (Bent 1929), but they also may feed on crustaceans, leeches, mollusks, small fish, and carrion (Oring et al. 1983). In the LDW, it was assumed that spotted sandpipers feed on amphipods and polychaetes in the intertidal mudflats along the LDW.

Exposure of spotted sandpiper via ingestion of prey was modeled using amphipod tissue data. The possibility that sandpiper could also ingest fish, mussels, or crab is addressed in the uncertainty assessment (Section A.7.3.2). Site-specific data on sediment ingestion by the spotted sandpiper were not available. However, EPA (1993b) summarized data on sediment ingestion by four other species of sandpipers that feed on mud-dwelling invertebrates. On a dry weight basis, the average sediment ingestion was 18% of the diet, with a range from 7.3 to 30%. It was assumed that sandpiper forage in intertidal areas because of their feeding habits, so only data from those areas were used in calculating the SWA sediment concentration.

Site Use Factor

Spotted sandpipers are a common bird in western Washington, and are known to nest along the LDW. They have been observed in the LDW from late June through September (Cordell et al. 1996), but have been known to overwinter locally (Paulson 1993). Nesting birds arrive in May and June. Canning et al. (1979) recorded seven spotted sandpiper nests located on Kellogg Island and at least three additional nest

sites were suspected on the island. Spotted sandpipers breed in open habitats along the margins of water bodies (Oring and Lank 1986).

A site use factor of 1.0 was assumed. Although spotted sandpiper are likely to forage within a subarea of the LDW based on a mean home range of 5 hectares (Miller and Miller 1948, as cited in Swarth 2002), site-specific information on spotted sandpiper use of the LDW was not available. A foraging range of about 1.5 km along the LDW was considered a reasonable estimate for spotted sandpiper and other similar shorebirds, such as dunlin, that are represented by sandpiper (Norman 2002b). The uncertainties associated with sandpiper site usage are discussed in the uncertainty assessment (Section A.7.3.2).

A.5.1.3.2 Great blue heron

Body Weight and Daily Food Consumption

The representative body weight of a female adult great blue heron is from Hartman (1961), as cited in EPA (1993b). The daily food consumption rate for females was calculated using an allometric equation for wading birds (Kushlan 1978):

$$DFC = 10^{0.966\log(BW)-0.64} \times \frac{0.001\ kg}{g} \qquad \textit{Equation 5-7}$$

Where:

DFC = daily food consumption rate (kg food/day, wet weight)
BW = wildlife species body weight (g)

The wet weight DFC rate was converted to dry weight assuming a moisture content in prey of 76%. This moisture content is based on the average of three English sole samples from the LDW; these were the only LDW whole-body fish samples analyzed for moisture content. The body weight and calculated DFC rate are shown in Table A-5-6.

Composition of Diet

Great blue heron feed in shallow water primarily on fish, but they also consume crustaceans, insects, amphibians, reptiles, and occasionally small mammals (Kushlan 1978; Butler 1993). Great blue heron hunt by sight and stalk or ambush their prey. They will also feed by probing, quickly moving their bills in and out of the water and substrate. Great blue heron feed on small fish that range in size from 8-33 cm (Kirkpatrick 1940; Alexander 1977; Hoffman 1978). Butler (1993) reported that shiner surfperch is a major food source for female and hatchling great blue heron and may be important for juvenile survival. The predominant prey items identified at the West Seattle colony were tails from sculpin (Krausmann 2002b). However, sculpin may not be representative of actual prey captured because the large lateral and dorsal spines make them more likely to be rejected by the heron chicks (Krausmann 2002b). In addition to perch and sculpin, heron may feed on other small fish in the LDW including juvenile salmonids and Pacific herring.

Shiner surfperch tissue data were used to calculate exposure to heron from ingestion of prey. The possibility that heron could also ingest substantial quantities of crab is addressed in the uncertainty assessment (Section A.7.3.2). Information on rates of sediment ingestion by heron was not available. It was assumed that a small amount of sediment would be ingested while probing for food in the substrate (estimated at 2% of the diet). It was assumed that great blue heron would consume sediment only from intertidal areas.

Site Use factor

A great blue heron colony of at least 37 active nests (presumably more than 74 birds) occupied a site in West Seattle in 1998 (Norman 2002a). In 1999, there were only 21 active nests. It is not known if the colony attempted to breed in 2000 or 2001, but no known successful nesting occurred. As of April 2002, the colony has remained empty (Norman 2002a). Other colonies are located 12 km south in Renton (Black River colony) and 12 km northwest near Salmon Bay (Kiwannis Ravine colony). Information presented in EPA (1993b) indicates that foraging grounds are generally close to breeding colonies, and that 15 to 20 km is the farthest great blue herons might regularly travel from the colony to the foraging area. Based on observations of individual birds from the West Seattle colony it was estimated that at least half of the birds from this colony used the LDW to forage, focusing primarily on the Kellogg Island area (Krausmann 2002a). Because the LDW could be their primary foraging ground, great blue herons are given a conservative site use factor of 1.0.

A.5.1.3.3 Bald eagle

Body weight and food consumption rate

The representative body weight for an adult female bald eagle (Table A-5-6) was obtained from Snyder and Wiley (1976, as cited in EPA 1993b). The food consumption rate was assumed to be 12% of the body weight on a wet weight basis, based on studies by Stalmaster and Gessaman (1982; presented in EPA 1993b) of free-flying eagles in Washington. The calculated daily food consumption rate for females (0.629 kg food ww/day) was converted to dry weight (0.151 kg food dw/day) using an average moisture content (76%) in whole-body fish from the LDW.

Composition of diet

Bald eagles are opportunistic foragers with site-specific food habits based on available prey species (Buehler 2000). Eagles consume dead and live fish, birds, and occasionally mammals. In most regions, bald eagles seek out aquatic habitats for foraging and prefer fish (Buehler 2000). Although eagles feed primarily on fish, a portion of their diet during winter months may include birds such as grebes, gulls, and waterfowl.

In four studies of bald eagles in western Washington and one in the Columbia River estuary, 85 to 92% of prey observed being foraged or delivered to the nest were fish, 6 to 15% were birds, and up to 3% were mammals (Knight et al. 1990; Watson et al. 1991; Sweeney et al. 1992; Watson et al. 1995; Watson and Pierce 1998). More recent

observations of 75 bald eagle territories throughout western Washington found lower overall use of fish (78%) and higher consumption of birds (19%) and mammals (3%) (Watson and Pierce 1998). Some of these studies conducted separate observations of prey remains at the base of nest trees, which showed lower proportions of fish and higher proportions of mammals. However, methods of data collection based on prey remains are suspected to be highly skewed toward items with hard bony structure (e.g., birds and mammals) and under-representative of soft-bodied items, especially fish (Buehler 2000).

In a study of prey remains at the base of nest trees throughout Puget Sound, 45 fish were identified; eight were rockfish, 10 were starry flounder, and the remainder included cod, pollock, hake, cabezon, red Irish lord, sculpins, surfperch, salmon, plainfin midshipman, and channel catfish (Knight et al. 1990). Ninety percent of 126 fish captured by eagles foraging at Hood Canal and Indian Island and classified by size were less than 30 cm long (Watson and Pierce 1998). Eagles have been reported to kill western grebes in the LDW during winter (Strand, personal observation as cited in King County [1999b]). Eagles also have been reported to prey on great blue heron chicks (Norman et al. 1989, as cited in King County [1999b]).

Any of the three types of fish collected in the LDW may be consumed by bald eagle, so they were assumed to constitute equal portions of the diet. Salmon were not analyzed for lead or mercury, so fish data with the higher concentration were used as a surrogate (English sole for lead and perch for mercury). Other prey of eagles higher on the food chain such as piscivorous birds may have higher COPC concentrations, although tissue data have not been collected. This potential data gap will be discussed in the uncertainty assessment (Section A.7.3.2). Data on sediment ingestion rates were not available, but it is likely that bald eagles consume a small amount of sediment while scavenging along the shoreline. Therefore, sediment ingestion was estimated to be 1% of the eagle's diet. It was assumed that only intertidal sediment would be ingested.

Site Use Factor

Five bald eagle nests within 8 km (5 mi) of the LDW were occupied in 1999 (King County 1999b). The closest nest is located in West Seattle within approximately 1.5 km (1 mi) of the LDW. One or two pairs of resident eagles may be found in the LDW vicinity during the summer (King County 1999b). Overwintering migrant eagles are routinely observed in the vicinity of the LDW from the beginning of October through late March. Home ranges of eight pairs of eagles from Squamish Harbor in Hood Canal ranged in size from 1.12 to 14.1 km^2, with core areas ranging between 0.27 and 4.2 km^2 (Watson et al. 1995). In nine bald eagle territories studied along Hood Canal from 1990 to 1997, the home range and core area sizes averaged 4.7 and 1.2 km^2, respectively (Watson and Pierce 1998). Because eagles may forage in nearby aquatic and terrestrial environments in addition to the LDW, the site usage factor is likely to be less than 1.0. However, definitive data to estimate this factor were lacking. Thus,

exposure to eagles was calculated using site-use factors of both 0.25 and 1.0 to present a range of potential exposure.

A.5.1.3.4 River otter

Body Weight and Food Consumption Rate

An adult body weight of 7.9 kg was assumed for a female river otter (Table A-5-6) based on a study by Melquist and Hornocker (1983), as cited in EPA (1993b). The daily food consumption rate was estimated as a function of the metabolic rate and the caloric content of the prey using the following equation:

$$DFC = \frac{FMR}{ME} \times \frac{0.001\,kg\,food}{g\,food} \qquad \textit{Equation 5-8}$$

Where:

DFC = daily food consumption rate (kg food/day dw)
FMR = free-living metabolic rate (kcal/day)
ME = average metabolizable energy of the total diet (kcal/g food dw)

The free-living metabolic rate (FMR) for females was calculated to be 1,180 kcal/day, using an equation developed by Nagy (1987, as cited in EPA 1993b) for placental mammals:

$$FMR\ (kcal/day) = 0.800 \times BW^{0.813} \qquad \textit{Equation 5-9}$$

where body weight is expressed in grams. The ME value used was that calculated by Nagy (1987; as cited in EPA 1993b) as a mean ME of 4.47 kcal/ g dw for mammals on a diet of fish. The final calculated food consumption rate for females was 0.264 kg/day (Table A-5-6).

Composition of Diet

River otters are opportunistic carnivores that take advantage of food that is most abundant and easiest to catch, although fish are their primary prey (EPA 1993b). River otters catch fish by diving and ambushing or chasing, and by digging in the substrate for invertebrates (Coulter et al. 1984). The slower moving fish such as suckers, carp, chubs, and bullheads are generally eaten most frequently (Wise et al. 1981; Kurta 1995). Studies in coastal southeast Alaska and British Columbia found that river otter feed primarily on sculpins, surfperch, and flatfish, with greenling, salmon, and rockfish making up lesser portions of the diet (Stenson et al. 1984; Larsen 1984). Other components of the river otter's diet include aquatic invertebrates (including crayfish, mussels and clams, and aquatic insects), frogs, snakes, and occasionally scavenged mammals and birds (Coulter et al. 1984). River otters generally consume fish ranging from 7.6 to 41 cm (Gilbert and Nancekivell 1982; Greer 1955, as cited by EPA 1993b), although Toweill (1974) found that many of the salmon preyed upon by river otters in western Oregon were up to an estimated 80 cm in length. These salmon were taken in coastal streams when fish enter the rivers to spawn. Local river otters feed largely on

fish, but will also feed on crabs and sometimes mussels and clams (Strand, personal observation, as cited in King County [1999b]).

The proportion of prey types consumed by river otter for this assessment was based on that reported by Larsen (1984) for southeastern Alaska. This study was used because it was the only study from the Pacific Northwest that reported remains in scat on a volume basis rather than as a frequency of occurrence. Larsen (1984) reported the following proportions of prey ingested by river otter: 86% fish, 10% crab, 2% invertebrates other than crab, 1% birds, and 1% mammals and plant material. Thus, for this assessment, otter are assumed to consume 86% fish, 10% crab, 2% mussels, and 2% sediment, because these are the dietary items for which chemical concentrations were available. Based on feeding habits of river otters documented in coastal southeast Alaska and British Columbia (Larsen 1984; Stenson et al. 1984), any of the three types of fish tissue for which chemistry data were available in the LDW might be consumed. Because no site-specific information was available on fish preference of river otter, it was assumed that perch, sole, and salmon were consumed in equal proportions of the 86% of their diet that is fish.

Data were not available from the literature on the amount of sediment consumed by river otters. A small amount of sediment is likely to be ingested while river otters forage on crab and benthic fish species, so the ingestion rate was estimated to be 2% of the diet. It was assumed that river otters incidentally ingest sediment from both intertidal and subtidal areas of the LDW.

Site Use Factor

Anecdotal information indicates that a river otter family lives year-round on Kellogg Island in the LDW, although otters have not been observed by Cordell during wildlife surveys (Cordell 2001b). River otters are almost exclusively aquatic and prefer food-rich habitats such as the lower portions of streams and rivers, estuaries, and lakes and tributaries that feed rivers (Tabor and Wight 1977; Mowbray et al. 1979). River otters range over an area sufficiently large enough for foraging and reproduction (Melquist and Dronkert 1987); however, they are typically found in a limited number of activity centers within their overall range. In streams, the river otter's home range can average 30 km (19 mi) (Melquist and Hornnocker 1983). At any given time, river otters generally occupy only a few kilometers of stream, but often move from one area to another (Nebraska Game and Parks Commission 2000). A radio-tracking study of relocated river otters was conducted as part of the New York State Department of Environmental Conservation river otter reintroduction program. This study showed that river otters' ranges were from 1.5 to 22.4 km long, with an average length of 10 km (6 mi) for individuals monitored in western New York State (Spinola et al. 1999, as cited in EPA 2000b).

No studies were found that document usage of the LDW by river otters. Because of the average 10 km linear length documented in the literature, and because the extent of the LDW study area is approximately 10 km, it was assumed river otter could

potentially consume prey and sediment solely from within the LDW. Therefore, it was conservatively assumed that the area use factor was equal to 1.0.

A.5.1.3.5 Harbor seal

Body Weight and Food Consumption Rate

A body weight for an adult female harbor seal of 76.5 kg was based on a study by Pitcher and Calkins (1979), as cited in EPA (1993b) (Table A-5-6). The food ingestion rate of female adult harbor seals was calculated with an allometric equation developed by Boulva and McLaren (1979, as cited in EPA 1993b) for harbor seals from eastern Canada, as follows:

$$DFC = 0.089 \times BW^{0.76} \qquad \textit{Equation 5-10}$$

Where:

DFC = daily food consumption rate (kg food/day ww)
BW = wildlife species body weight (kg)

The calculated wet weight DFC of 2.40 kg food/day for females was converted to a dry weight value of 0.577 kg food/day using the average moisture content (76%) in whole-body fish from the LDW.

Composition of Diet

Harbor seals are opportunistic feeders, selecting prey based on availability and ease of capture (Pitcher and Calkins 1979; Pitcher 1980; Schaffer 1989). Their diet can vary seasonally with local abundance and includes bottom-dwelling fishes, invertebrates such as octopus and squid, and species that congregate for spawning (Pitcher and Calkins 1979; Everitt et al. 1981; Lowry and Frost 1981; Roffe and Mate 1984). In Washington, the most important prey include Pacific whiting, tomcod, walleye pollock, flatfishes, Pacific herring, shiner surfperch, plainfin midshipman, and sculpins (NMFS 1997). Fish consumed were generally between 4 and 28 cm in length (Brown and Mate 1983).

Harbor seals may prey on salmonids during adult upriver spawning migrations or juvenile downriver outmigrations in the LDW, although site-specific data were not available on the dietary importance of migrating salmon to local seal populations. Predation on pink salmon in the fall, steelhead in the winter, and chinook salmon in the spring has been reported in Puget Sound (Everitt et al. 1981). Harbor seal food habits in Washington have been evaluated by observing scat for the occurrence of prey species. No data have been collected in Elliott Bay, but the highest percentage of samples in which salmonid remains were found was 50%, reported in seals caught incidentally in the salmon gillnet fishery in Grays Harbor (NMFS 1997). In other areas of Washington, salmonid remains were found in zero to 28% of scat samples. Kaczynski and Palmisano (1992, as cited in NMFS 1997) estimated the annual consumption of salmonids in Oregon to be 10.8% of the total biomass that seals consume. Because site-specific information was not available on the amount of each

type of prey consumed, it is assumed that salmon, sole, and perch are ingested in equal proportions. Data on sediment ingestion were not available, but it is likely that a small amount of sediment is incidentally consumed while foraging on bottom fish. Therefore, a 2% rate of ingestion was assumed. The lack of specific information on extent of foraging in the LDW and types of prey consumed are discussed in the uncertainty assessment (Section A.7.3.2).

Site Use Factor

Harbor seals are commonly seen in Elliott Bay and occasionally enter the LDW (Kenney 1982). Harbor seals have been shown to forage over large areas ranging from 5 km (3 mi) (Stewart et al. 1989) to 55 km (34 mi) (Beach et al. 1985). In Puget Sound, seals generally forage within 8 to 13 km (5 to 8 mi) of their haulout areas established as pupping sites (Jeffries 2001). The closest pupping site is located at Blakely Rocks off the southeast end of Bainbridge Island, approximately 12 km (7 mi) from the LDW. Site-specific information on harbor seal usage of the LDW is limited. The WDFW observed harbor seals infrequently in the LDW during an intensive survey conducted from December 1998 to June 1999 (WDFW 1999). This survey monitored the waterway for the presence of sea lions and seals up to the 16th Avenue South Bridge for a total of 307 hours on 52 days. Seals were observed on 17 occasions, and were most frequently seen north of the 1st Avenue South Bridge. While seals have been observed in Elliott Bay and may use log booms to haul out, they are not known to aggregate in large numbers (Jeffries 2001). Fish from the LDW may constitute a small portion of the seal's diet based on the presence of other areas containing prey species within the foraging radius of the seal population on Bainbridge Island. However, the LDW may be a preferential feeding area during chinook salmon outmigration from March through August. In the Columbia River, salmonids appear to be targeted as prey by seals in the spring and fall when they are abundant and available in the river (NMFS 1997).

Data from the WDFW survey discussed above were used to establish a site use factor for risk calculations. The following conservative assumptions were used: 1) the same seal was observed on all 17 occasions out of 52 days of observation, 2) the seal obtained all its food for that day in the LDW, and 3) site usage from December through June accurately represent usage during other times of the year. Based on these assumptions, the SUF was equal to 17/52 or 0.33. This same observation is supported for sea lions, which were observed on 16 occasions during the same period of observation.

A.5.1.4 Exposure results

Exposure dose calculations were made using Equations 5-1 and 5-2. Tissue and sediment data used in these equations are shown in Tables A-5-3 and A-5-4. Exposure factor values are shown in Table A-5-6. With the exception of copper and zinc in sandpipers, female body weights were used for all ROC/COPC pairs, because the TRVs presented in Section A.5.2 were based primarily on reproductive effects in

females. The avian TRVs for copper and zinc were based on other non-gender-specific effects, so the average of male and female body weights was used instead.

Table A-5-7 presents the input values and calculation results for prey concentrations for each ROC/COPC pair. Table A-5-8 presents the calculated exposure doses on a daily basis adjusted for body weight, along with the input values.

Table A-5-7. Concentrations of COPCs in food for each ROC/COPC pair (using Equation 5-2)

Chemical	Concentration in Prey[a] (mg/kg dw)						Fraction in Diet						Concentration in Food (mg/kg dw)
	Perch	Sole	Salmon	Amphipod	Crab[b]	Mussel	Perch	Sole	Salmon	Amphipod	Crab	Mussel	
Sandpiper													
PCBs				2.18						1.0			2.18
Copper				166						1.0			166
Lead				41.4						1.0			41.4
Zinc				132						1.0			132
BEHP				2.62						1.0			2.62
Great blue heron													
PCBs	2.57						1.0						2.57
Lead	0.729						1.0						0.729
Mercury	0.367						1.0						0.367
Bald eagle													
PCBs	2.57	9.15	0.367				0.333	0.333	0.333				4.12
Lead	0.729	0.88	0.88[c]				0.333	0.333	0.333				0.829
Mercury	0.367	0.338	0.367[c]				0.333	0.333	0.333				0.357
River otter													
PCBs	2.57	9.15	0.367		1.79	0.179	0.293	0.293	0.293		0.1	0.02	3.81
Arsenic	5.79	25.8	25.8[c]		60.3	3.78	0.293	0.293	0.293		0.1	0.02	22.9
Lead	0.729	0.88	0.88[c]		1.20	2.20	0.293	0.293	0.293		0.1	0.02	0.893
Harbor seal													
PCBs	2.57	9.15	0.367				0.333	0.333	0.333				4.12

[a] Dry weights calculated using measured moisture content with the exception of perch, salmon, and mussel, which were not analyzed for moisture content. For those tissue types, the average moisture content in English sole was used (76%).

[b] Concentrations reflect a weighted value using 85% edible meat concentration and 15% hepatopancreas concentration.

[c] Metals data were not available for whole body juvenile salmon, so the highest concentration from perch or sole data were used as surrogates (lead and arsenic from English sole, and mercury from perch).

Table A-5-8. Calculated exposure doses for each ROC/COPC pair (using Equation 5-1)

Chemical	Food Consumption Rate (kg/day dw)	Sediment Consumption Rate (kg/day dw)	Concentration in Food (mg/kg dw)	Concentration in Sediment (mg/kg dw)	Site Use Factor	Body Weight[a] (kg ww)	Exposure Dose (mg/kg bw/day
Sandpiper							
PCBs	0.0074	0.0013	2.18	0.73	1	0.0471	0.363
Copper	0.0067	0.0012	166	48	1	0.0425	27.5
Lead	0.0074	0.0013	41.4	61	1	0.0471	8.23
Zinc	0.0067	0.0012	132	110	1	0.0425	23.9
BEHP	0.0074	0.0013	2.62	0.27	1	0.0471	0.419
Great blue heron							
PCBs	0.0931	0.0019	2.57	0.73	1	2.2	0.109
Lead	0.0931	0.0019	0.729	61	1	2.2	0.0825
Mercury	0.0931	0.0019	0.367	0.12	1	2.2	0.0156
Bald eagle[b]							
PCBs (SUF=0.25)	0.151	0.0015	4.12	0.73	0.25	5.24	0.0298
PCBs (SUF=1)	0.151	0.0015	4.12	0.73	1	5.24	0.119
Lead (SUF=0.25)	0.151	0.0015	0.829	61	0.25	5.24	0.0104
Lead (SUF=1)	0.151	0.0015	0.829	61	1	5.24	0.0415
Mercury (SUF=0.25)	0.151	0.0015	0.357	0.12	0.25	5.24	0.0026
Mercury (SUF=1)	0.151	0.0015	0.357	0.12	1	5.24	0.0103
River otter							
PCBs	0.264	0.0053	3.81	0.37	1	7.9	0.128
Arsenic	0.264	0.0053	22.9	12	1	7.9	0.774
Lead	0.264	0.0053	0.89	48	1	7.9	0.0619
Harbor seal							
PCBs	0.577	0.0115	4.12	0.37	0.33	76.5	0.0103

[a] With the exception of copper and zinc for sandpiper, female body weights and food consumption rates were used for all ROC/COPC pairs because all other TRVs were based on reproductive effects from female exposure. Mean male and female weights and food consumption rates were used for sandpipers for the assessment of copper and zinc because the TRVs for these chemicals were based on other non-gender-specific effects.

[b] Calculations for eagle were made using two site use factors to show a range in potential exposure dose.

A.5.2 Effects Assessment

In the effects assessment the toxicity literature for COPCs identified in the problem formulation was reviewed and levels that represent threshold effect concentrations of COPCs for the ROCs were selected. In the risk characterization (Section A.7.3), these thresholds are compared to the exposure doses or PCB concentrations in heron eggs presented in Section A.5.1 (Table A-5-8) to estimate risk.

To develop threshold concentrations, the toxicity literature was searched and single-chemical toxicity data for birds and mammals were compiled. Toxicity studies published in the scientific literature were found from review sources or electronic databases (i.e., BIOSIS, TERRETOX, Science Citation Index, Cal/EPA database). Review sources used to obtain effects data included the following:

- U.S. Fish and Wildlife Service Contaminant Review series (Eisler)
- Agency for Toxic Substances and Disease Registry
- Oak Ridge National Laboratory database (Sample et al. 1996)

Toxicity endpoints identified from laboratory data include both the no observed adverse effect level (NOAEL; the highest dose or egg concentration at which no effect was observed) and lowest observed adverse effect level (LOAEL; the lowest dose or egg concentration at which an effect was observed).[89] The most appropriate NOAEL and LOAEL were chosen for each ROC/COPC pair to be used as toxicity reference values (TRVs), based on considerations discussed in the following paragraph. These TRVs are used in the risk characterization (Section A.7.3.1) as benchmark values for comparison to estimated exposure doses or egg concentrations presented in Section A.5.1. The TRV based on the NOAEL represents the threshold level of COPCs, below which adverse effects would not be expected for the endpoint studied. The TRV based on the LOAEL represents the level above which an effect would be expected.

Data used to develop TRVs were extracted from original sources to verify effect levels, quality of study design, magnitude of dose, and other study parameters. Studies were not considered unless negative control groups were included and results were evaluated statistically to identify significant differences from control values. NOAELs and LOAELs considered as TRVs for dietary exposure were prioritized using the following guidelines:

- Exposure was chronic, preferably lasting more than one year for mammals and more than 10 weeks for birds (Sample et al. 1996), or conducted during a critical life stage such as reproduction, gestation, or development
- Birds tested were non-domesticated wildlife species
- The chemical form was bioavailable and similar to the form potentially ingested at the LDW area
- Preferred dose was through ingestion of food rather than through drinking water ingestion, gavage, intraperitoneal injection, or oral intubation

In most cases, the toxicity literature presented data only as a concentration in food, so these values were converted to a daily dose (mg/kg bw/day) using the animal's body

[89] Although the fish benchmarks are expressed as NOECs and LOECs, the terms NOAEL and LOAEL are used for wildlife benchmarks because they represent body-weight-normalized dose levels rather than tissue concentrations.

weight and ingestion rate. The assumptions used in converting the dietary concentration to a dose are footnoted in tables presenting toxicity data.[90]

If the low-effect dose chosen for a TRV did not have an associated no-effect dose, the no-effect dose from another appropriate study was used. If an appropriate no-effect dose was not available, the LOAEL was divided by an uncertainty factor of 10, following methodology of Sample et al. (1996).

Effect endpoints considered in the literature were those related to the assessment endpoints of survival, growth, and reproduction. For PCBs, toxicity data on survival and growth were not compiled, because those effect concentrations were substantially higher than those available for reproductive endpoints. The PCB TRVs based on reproduction will, therefore, be protective of other potential effects noted at higher concentrations.

The following sections present the COPC-specific rationale for TRVs chosen for mammals and birds.

A.5.2.1 Dietary TRVs for birds

The COPCs identified for birds in the problem formulation were PCBs, copper, lead, mercury, zinc, and BEHP.[91] This section discusses general toxicity information for each of these COPCs, compares toxicity studies to guidelines outlined in Section A.5.2, and selects avian TRVs for each COPC.

A significant body of literature exists that documents effects of dietary exposure on poultry. However, the domestic chicken is a poor surrogate for wildlife receptors for reproductive toxicants. Chickens have been bred specifically to have unnaturally high egg-laying rates, exceeding one egg every two days. Even with a significant reduction in the baseline egg production, a laying hen may still have an egg production rate much greater than any wild avian species. Thus, extrapolating an apparent reproductive "effect threshold" in domestic chickens to wildlife receptors is questionable, because of differences in reproductive physiology. Similar concerns apply to Japanese quail, a species also bred specifically for egg production. Because of these considerations, this effects assessment does not use chicken or Japanese quail data for developing TRVs based on reproductive effects, unless there was a lack of other more appropriate studies.

---

[90] There may be differences in some TRVs calculated in this document when compared to those calculated by King County (1999c) using the same studies and endpoints, because different body weights and ingestion rates may have been used in the calculations. King County did not document body weights and ingestion rates used in their calculations, so is was not possible to compare the TRV derivations.

[91] COPCs for sandpiper were PCBs, BEHP, Cu, Pb, and Zn; the COPCs for heron and eagle were PCBs, Hg, and Pb (see Table A-5-1).

A.5.2.1.1 PCBs

Effects of avian exposure to dietary PCBs include disruption of normal patterns of growth, reproduction, metabolism, and behavior (Eisler 1986b). The most sensitive effects are related to reproduction, and include egg production, fertility, and hatching success. Of the laboratory species used to examine reproductive endpoints, chickens have been found to be the most sensitive to PCBs (Kennedy et al. 1996). However, because of concerns with poultry laboratory studies, only data from wildlife laboratory studies (Table A-5-9) were considered in choosing an avian PCB TRV. This approach is supported by the findings of an EPA-sponsored peer review panel charged with reviewing an ERA for the Hudson River. This panel recently evaluated use of PCB TRVs derived from chicken studies to assess risk to wild birds. The reviewers considered PCB TRVs developed from chicken studies to be "unrealistically low and excessively conservative" and found that "using the chicken as a representative species for wild birds was not defensible" (EPA 2000b). The use of chicken reproductive toxicity data to assess risk to wildlife species should thus be considered protective, but it is not likely to predict risk accurately. These data were therefore not used.

Methods and results of the three avian wildlife studies that presented PCB effect concentrations (Table A-5-9; Peakall et al. [1972] with ringed turtledove; Dahlgren et al. [1972] with ring-necked pheasant; Haseltine and Prouty [1980] with mallard) were compared using the guidelines for choosing a TRV presented in Section A.5.2, and the following observations were made:

- In all studies, exposure was chronic and occurred during reproduction.
- All of the birds tested were non-domesticated wildlife species.
- Aroclor 1254, the mixture of PCBs used by Peakall et al. (1972) and Dahlgren et al. (1972), was detected in prey tissue from the LDW, whereas Aroclor 1242, used by Haseltine and Prouty (1980), was not detected in prey tissue.
- Exposure in all three studies was through food ingestion.

Based on the above comparison, the studies with ringed turtledove and ring-necked pheasant (also the two with the lowest effect levels) were most appropriate to use as TRVs. Because of uncertainty in sensitivity differences between the two species, the lower effects value of the two studies was used (a LOAEL of 0.94 mg/kg bw/day measured in ringed turtledove by Peakall et al. 1972). The lowest and most appropriate PCB NOAEL for reproductive effects in birds identified was based on the two-year study that McLane and Hughes (1980) conducted to examine the reproductive effects of PCBs on screech owls (NOAEL of 0.41 mg/kg bw/day).

Table A-5-9. Laboratory data for the effects of dietary PCBs on birds

Test Organism	Analyte	Exposure route	Exposure duration	LOAEL (mg/kg bw/d)	NOAEL (mg/kg bw/d)	Effect Concentration (mg/kg ww)	Exposure Concentrations (mg/kg ww)	Effect Endpoint	Reference
Ringed turtle-dove	Aroclor 1254	Diet	2 generations	0.94[a]	nd	10	0 and 10	Hatching success in second generation	Peakall et al. 1972
Ring-necked pheasant	Aroclor 1254	Diet, in gelatin capsules	Once per week for 16 weeks	1.8[b]	nd	12.5 mg/bird /week	0, 12.5, 50 (mg/bird/week)	Egg hatchability	Dahlgren et al. 1972
Mallard	Aroclor 1242	Diet	12 weeks	15[c]	nd	150	0 and 150	Eggshell thinning	Haseltine and Prouty 1980
Screech owl	Aroclor 1248	Diet	2 generations	nd	0.41	ne	0 and 3	Eggshell thickness, egg production, hatching success, fledging success	McLane and Hughes 1980
Mallard	Aroclor 1254	Diet	4 months	nd	3.9	ne	0 and 39	Egg production, eggshell thinning	Risebrough and Anderson 1975
Mallard	Aroclor 1254	Diet	Approx. 1 month	nd	7	ne	0 and 25	Reproductive success	Custer and Heinz 1980

nd – No data available

ne – No effect observed at any dose

a Assuming a body weight of 0.155 kg (Sample et al. 1996) and an ingestion rate of 0.0146 kg/day ww. The dry weight ingestion rate was calculated using an allometric equation for non-passerines (Nagy 1987, as cited in EPA 1993b), and adjusted to wet weight assuming a moisture content of 9% in seeds (EPA 1993b).

b Assuming a body weight of 1 kg (Sample et al. 1996).

c Assuming a body weight of 1 kg and ingestion rate of 0.1 kg/day ww (EPA 1993b).

A.5.2.1.2 Copper

Avian wildlife species have not been used in the laboratory to evaluate toxicity from dietary copper. The only available studies from which to identify a copper TRV addressed growth and survival of chickens (Table A-5-10; Mehring et al. 1960; Poupoulis and Jensen 1976; Smith 1969). These studies showed decreased growth at dietary concentrations ranging from 350 to 749 mg/kg ww. Concentrations of 749 mg/kg ww in the diet for 10 weeks resulted in increased mortality of chicks (Mehring et al. 1960).

When the methods and results of the three copper studies conducted with chickens were compared using the guidelines for choosing NOAELs and LOAELs presented in Section A.5.2, the primary difference among studies was length of exposure. The study by Mehring et al. (1960) was the only study conducted for at least 10 weeks; the other exposures were from 3 to 4 weeks. Therefore, the LOAEL (62 mg/kg bw/day) and NOAEL (47 mg/kg bw/day) from Mehring et al. (1960) were selected as copper TRVs.

A.5.2.1.3 Lead

Extensive data have been collected on the acute effects of lead poisoning in birds as a result of lead shot ingestion. Numerous effects have been observed that lead to death, as well as damage to the nervous system, muscular paralysis, kidney and liver damage, internal lesions, enlarged gall bladder, anemia, reduced brain weight, and abnormal skeletal development (Eisler 1988a). Fewer studies have been conducted on chronic effects of dietary exposure to lead in the laboratory. The most sensitive effects observed in laboratory studies (Table A-5-11) were decreased egg production after 4 to 5 weeks of exposure in chicken and Japanese quail, observed at LOAELs from 0.2 to 3.3 mg/kg bw/day. As with chickens, Japanese quail are domestic birds bred specifically for high rates of egg production, so application of these results to potential reproductive effects in wildlife is questionable. Egg hatchability was decreased at a higher LOAEL of 20 mg/kg bw/day in Japanese quail.

Table A-5-10. Laboratory data for the effects of dietary copper on birds

Test Organism	Analyte	Exposure Route	Exposure Duration	LOAEL (mg/kg bw/d)	NOAEL (mg/kg bw/d)	Effect Concentration (mg/kg ww)	Exposure Concentrations (mg/kg ww)	Effect Endpoint	Reference
Chicks, day-old	Copper sulfate	Diet	25 days	29[a]	16[a]	350	10, 100, 200, and 350	Growth	Smith 1969
Chicks	Copper sulfate	Diet	4 weeks	41[a]	21[a]	500	250, 500, and 1,000	Growth and gizzard erosion	Poupoulis and Jensen 1976
Chicks	Copper oxide	Diet	10 weeks	62[a]	47[a]	749	26, 37, 52, 74, 104, 147, 208, 294, 403, 570, 749, and 1180	Growth, mortality	Mehring et al. 1960

a Assuming a body weight of 0.534 kg and ingestion rate of 0.044 kg/day ww (EPA 1993b).

Table A-5-11. Laboratory data for the effects of dietary lead on birds

Test Organism	Analyte	Exposure Route	Exposure Duration	LOAEL (mg/kg bw/d)	NOAEL (mg/kg bw/d)	Effect Concentration (mg/kg ww)	Exposure Concentrations (mg/kg ww)	Effect Endpoint	Reference
Japanese quail, from day of hatching	Lead acetate	Diet	5 weeks	0.20[a]	nd	1	0, 1, 10, and 100	Egg production	Edens and Garlich 1983; Edens et al. 1976
Japanese quail, 6 weeks old	Lead acetate	Diet	5 weeks	2.0[a]	0.20[a]	10	0, 1, 10, and 100	Egg production	Edens and Garlich 1983
Leghorn chickens	Lead acetate	Diet	4 weeks	3.3[b]	1.65[b]	50	0, 25, and 50	Egg production	Edens and Garlich 1983
Japanese quail	Lead acetate	Diet	12 weeks	20[a]	2.0[a]	100	0, 1, 10, 100, and 1000	Egg hatchability	Edens et al. 1976
American kestrel nestlings	Metallic lead powder	Oral intubation	10 days	125	25	125 mg/kg bw/day	0, 25, 125, and 625 mg/kg bw/day	Growth	Hoffman et al. 1985
Mallards, first-year	Lead nitrate	Diet	12 weeks	nd	0.10[c]	ne	0, 1, 5, and 25 mg/kg food	Mortality, pathologic lesions	Finley et al. 1976
American kestrel	Metallic lead powder	Diet	5-7 months	nd	3.85[d]	ne	0, 5, and 50 mg/kg/food	Mortality, fertility, egg production, eggshell thinning	Pattee 1984
Ringed turtledoves	Lead acetate	Oral intubation	7 days	nd	75	ne	0, 25, 50, and 75 mg/kg bw/day	Growth, ALAD activity	Kendall and Scanlon 1982

nd – No data available

ne – No effect observed at any dose

a Using a body weight of 0.155 kg and ingestion rate of 0.031 kg/day ww (measured in Edens and Garlich 1983).

b Using a body weight of 1.84 kg and ingestion rate of 0.121 kg/day ww (measured in Edens and Garlich 1983).

c Using a body weight of 1 kg and ingestion rate of 0.100 kg/day ww (Heinz et al. 1989).

d Using a body weight of 0.13 kg (measured in study) and ingestion rate of 0.01 kg/day ww (Sample et al. 1996).

Methods and results of the lead studies presented in Table A-5-10 were compared using the guidelines for choosing a TRV, and the following observations were made:

- All studies were conducted for at least 10 weeks or during reproduction except two studies with American kestrel (Hoffman et al. 1985) and ringed turtledoves (Kendall and Scanlon 1982).
- The only non-domesticated wildlife species tested were American kestrel, mallard, and ringed turtledove.
- Two studies with American kestrel used metallic lead powder (Hoffman et al. 1985; Pattee 1984), a form of lead not likely representative of the LDW site.
- All studies involved dietary ingestion except two that used oral intubation with kestrels (Hoffman et al. 1985) and ringed turtledoves (Kendall and Scanlon 1982).

Based on the above comparison, effects values from the Japanese quail study (Edens et al. 1976) with an endpoint of egg hatchability rather than egg production were considered most appropriate. The NOAEL and LOAEL values from this study were 2.0 and 20 mg/kg bw/day, respectively.

A.5.2.1.4 Mercury

Laboratory studies have been conducted using a variety of wildlife species to evaluate the toxicity of mercury. When reviewing the toxicity literature for mercury, only forms of mercury relevant to the LDW were considered. Acceptable forms included inorganic mercury salts, such as mercuric chloride, as well as organic forms, such as methyl mercury chloride and dimethylmercury. Mercury-containing fungicides (e.g., Ceresan, methyl mercury dicyandiamide) were not considered relevant because these forms of the chemical are not natural nor are they likely to be present in the LDW. The toxicity of these fungicidal formulations is likely highly influenced by the attached anions that are intended to enhance the toxicity of the fungicide due to the additive effects of these non-mercury components. As a result, a few laboratory bird studies were not considered for TRV evaluation.

As presented in Table A-5-12, observed adverse effects include decreased growth and appetite in developing great egrets (Spalding et al. 2000), eggshell thinning in Japanese quail (Stoewsand et al. 1971), and mortality in zebra finch and bobwhite (Spann et al. 1986; Scheuhammer 1988). Based on a comparison with the guidelines for choosing a TRV:

- All studies were conducted for at least 10 weeks except for one study with bobwhite that was conducted during developmental stages.
- Two studies used species in the same taxonomic order as ROCs; American kestrel is in the same order as bald eagle (Falconiformes) and great egret is in the same order and family as great blue heron (Ciconiiformes, Ardeidae).

- All studies except the Japanese quail study used methylmercury, the most toxic form of mercury and the form of mercury most prevalent in fish. The quail study used an inorganic mercury salt (mercuric chloride).
- All studies involved dietary ingestion.

Thus, the most appropriate effects data for great blue heron were from the study with great egret (Spalding et al. 2000), which also had the lowest LOAEL (0.091 mg/kg bw/day) of the studies. The American kestrel study (Peakall and Lincer 1972), which used the species most similar to eagle, resulted in no effect on eggshell thinning at a dose of 0.77 mg/kg bw/day. It is possible that the growth endpoint in developing kestrels could be more sensitive than eggshell thinning, although the growth endpoint was not measured. Therefore, the LOAEL from the egret study was selected for characterizing risk to bald eagle as well as heron. No NOAEL was reported for the egret study, so it was derived using a factor of 10. The resulting TRVs for mercury were 0.0091 and 0.091 mg/kg bw/day for the NOAEL and LOAEL, respectively.

A.5.2.1.5 Zinc

Most laboratory studies on zinc toxicity have been conducted with domestic birds such as chickens; only one wildlife study, using mallards, was found in the literature. Observed effects of zinc exposure on ducks and chickens in laboratory studies include reduced food intake and egg production, cessation of egg laying, weight loss, leg paralysis, pancreatic histopathology, and mortality (Eisler 1993). Adverse effects may also be observed if zinc is deficient in the diet because zinc is a nutrient essential for normal growth, development, and function. However, effects from zinc deficiency are generally noted at concentrations below 120 mg/kg ww in food (Eisler 1993). Table A-5-13 summarizes results from several laboratory studies considered in developing the avian TRV for zinc.

Methods and results from laboratory studies in Table A-5-13 were compared to the guidelines for choosing TRVs in Section A.5.2, as follows:

- Only one study (Stahl et al. 1990) was conducted for more than 10 weeks or during a reproductive life stage; however, no effects were found in this study.
- None of the species tested were in the same taxonomic order as the ROCs.
- Bioavailable forms of zinc were used in all studies.
- The form of exposure was through dietary ingestion in all studies.

The most appropriate studies to use for TRVs were Roberson and Schaible (1960) and Gasaway and Buss (1972), although neither study was conducted for more than 10 weeks or during a critical life stage. The lowest effect doses, from Roberson and Schaible (1960), were chosen as TRVs. The resulting NOAEL and LOAEL doses were 82 and 123 mg/kg bw/day, respectively.

Table A-5-12. Laboratory data for the effects of dietary mercury on birds

Test Organism	Analyte	Exposure Route	Exposure Duration	LOAEL (mg/kg bw/d)	NOAEL (mg/kg bw/d)	Effect Concentration (mg/kg ww)	Exposure Concentrations (mg/kg ww)	Effect Endpoint	Reference
Great egret, one day old	Methylmercury chloride	diet	14 weeks	0.091[a]	nd	0.5	0, 0.5, and 5	Growth	Spalding et al. 2000
Zebra finch	Methylmercury chloride	diet	76 days	0.30[b]	0.1[b]	1	0, 0.2, 0.5, and 1[c]	Survival	Scheuhammer 1988
Mallard	Methylmercury chloride	diet	>60 days	nd	0.5[d]	ne	0 and 5	Eggshell thickness	Heinz 1980
American kestrel	Dimethyl mercury	diet	3 months	nd	0.77[e]	ne	0 and 10	Eggshell thickness	Peakall and Lincer 1972
Japanese quail, one day old	Mercuric chloride	diet	10 weeks	1.6[f]	0.80[f]	8	1, 1, 2, 4, and 8	Eggshell thickness	Stoewsand et al. 1971
Northern bobwhite, 12-day old	Methylmercury chloride	diet	6 weeks	1.6[g]	0.43[g]	20	0, 5.4, and 20	Survival	Spann et al. 1986

nd – no data available

ne – No effect observed at any dose

[a] Using a body weight of 1.02 kg and ingestion rate of 0.185 kg/day ww (Arizona Game and Fish 2002; Kushlan 1978).

[b] Using a body weight of 0.012 kg and ingestion rate of 0.0036 kg/day ww (Dunning 1993; Sample et al. 1996).

[c] Converted from dry weight using a moisture content of 80% (Heinz 1979; percent moisture in dry duck mash).

[d] Using a body weight of 1 kg and ingestion rate of 0.100 kg/day ww (Heinz et al. 1989).

[e] Using a body weight of 0.13 kg and ingestion rate of 0.01 kg/day ww (Pattee 1984; Sample et al. 1996).

[f] Using a body weight of 0.155 kg and ingestion rate of 0.031 kg/day ww (Edens and Garlich 1983).

[g] Using a body weight of 0.19 kg and ingestion rate of 0.015 kg/day ww (EPA 1993b).

Table A-5-13. Laboratory data for the effects of dietary zinc on birds

Test Organism	Analyte	Exposure Route	Exposure Duration	LOAEL (mg/kg bw/d)	NOAEL (mg/kg bw/d)	Effect Concentration (mg/kg ww)	Exposure Concentrations (mg/kg ww)	Effect Endpoint	Reference
White rock chicks	Zinc oxide, zinc sulfate, or zinc carbonate	Diet	5 weeks	123[a]	82[a]	1,500	0, 1,000, 1,500, 2,000, and 3,000	Growth	Roberson and Schaible 1960
White leghorn hens	Zinc sulfate	Diet and supplements	44 weeks	nd	134[b]	ne	0, 48, 228, and 2,028	Egg hatchability	Stahl et al. 1990
Mallard (7 wk old)	Zinc carbonate	Diet	60 days	300[c]	nd	3,000	0, 3,000, 6,000, 9,000, and 12,000	Mortality, leg paralysis	Gasaway 1972
Hubbard broiler chicks	Zinc acetate	Diet	5 weeks	656[a]	328[a]	8,000	0, 1,000, 2,000, 4,000, 8,000, and 16,000	Mortality, reduced growth	Oh et al. 1979

nd – No data available

ne – No effect observed at any dose

[a] Using a body weight of 0.534 kg and ingestion rate of 0.044 kg/day ww (Sample et al. 1996).

[b] Using a body weight of 1.9 kg and ingestion rate of 0.125 kg/day ww (measured in study control hens).

[c] Using a body weight of 1 kg and ingestion rate of 0.100 kg/day ww (Heinz et al. 1989).

A.5.2.1.6 BEHP

Limited data were available for effects of BEHP on birds (Table A-5-14). Only one study was found in the scientific literature, in which effects were noted, but these effects were observed at a relatively high dose (350 mg/kg bw/day) in chickens, and no lower doses were tested (Ishida et al. 1982). Two other studies did not find growth or reproductive effects at the highest concentrations tested: 1.1 mg/kg/day for turtledoves and 5.1 mg/kg/day for starling (Peakall 1974; O'Shea and Stafford 1980). The starling study (O'Shea and Stafford 1980) did not include reproductive endpoints and was not conducted for more than 10 weeks. In the absence of other data, the single LOAEL of 350 mg/kg/day was selected from Ishida et al. (1982) along with the highest NOAEL of 5.1 mg/kg/day from O'Shea and Stafford (1980).

A.5.2.2 Egg TRVs for birds

Great blue heron eggs collected from near the LDW were analyzed for PCB congeners, so egg-residue-based TRVs are presented for total PCBs and TCDD toxic equivalents (TEQs) in this section. Four laboratory studies using wildlife species were considered appropriate for relating total PCB concentrations in eggs to adverse effects (Table A-5-15). These studies exposed adult birds to Aroclor mixtures in their diet and measured effects in first or second generation on hatching success, eggshell thickness, egg production, fledging success, or egg laying (Peakall et al. 1972; Peakall and Peakall 1973; Haseltine and Prouty 1980; McLane and Hughes 1980; Fernie et al. 2001). All studies involved chronic exposure. Although wildlife species were used, none were in the same taxonomic order as great blue heron. The lowest of the three observed effect concentrations for total PCBs (16 mg/kg ww in egg), which measured hatching success in the second generation of ringed turtledoves, was selected as the LOAEL (Peakall et al. 1972; Peakall and Peakall 1973). Because the LOAEL was unbounded, the only no-effect concentration—from a screech owl study (McLane and Hughes 1980)—was chosen as the NOAEL (7.1 mg/kg ww).

There were several laboratory studies with chickens and PCB effect concentrations in eggs (Britton and Huston 1973; Tumasonis et al. 1973; Scott 1977), but as discussed previously, it is not realistic to use chickens as a surrogate for wildlife species. The egg PCB effect concentrations for chickens ranged from 1.5 to 4.0 mg/kg ww.

The only laboratory studies for PCB congeners or dioxins involving wildlife and TEQs were conducted using chemical injection into the egg. While this is not an environmentally representative exposure route, results from studies using both egg injection and feeding methods compare favorably (Hoffman et al. 1996). Data were available for six wildlife species dosed with TCDD, PCB 126, or PCB 77[92] (Table A-5-16). Great blue heron was one of the species tested, with an unbounded

[92] The following TEFs were used in the toxicity studies to convert the effect concentrations to TEQs: 1 for TCDD, 0.1 for PCB 126 and 0.05 for PCB 77.

Table A-5-14. Laboratory data for the effects of dietary BEHP on birds

Test Organism	Analyte	Exposure Route	Exposure Duration	LOAEL (mg/kg bw/d)	NOAEL (mg/kg bw/d)	Effect Concentration (mg/kg ww)	Exposure Concentrations (mg/kg ww)	Effect Endpoint	Reference
Ringed turtle - dove	BEHP	Diet	4 weeks	nd	1.11	ne	0 and 10	Eggshell thickness	Peakall 1974
European starling	BEHP	Diet	30 days	nd	5.1	ne	0, 25, and 250	Growth, food consumption	O'Shea and Stafford 1980
Chicken	BEHP	Diet	230 days	350	nd	5,000	5,000 and 10,000	Cessation of egg laying, abnormal ovaries	Ishida et al. 1982

nd – No data available

ne – No effect observed at any dose

Table A-5-15. Laboratory data for the effects of PCBs in eggs on birds

Test Organism	Analyte	Exposure Route	Exposure Duration	LOAEL Egg Concentration (mg/kg ww)	NOAEL Egg Concentration (mg/kg ww)	Effect Endpoint	Reference
Ringed turtledove	Aroclor 1254	Diet	2 generations	16	nd	Hatching success in second generation	Peakall et al. 1972; Peakall and Peakall 1973
American kestrel	Aroclor 1248:1254: 1260 mixture	Diet	100 days in first generation	34	nd	Egg laying in second generation	Fernie et al. 2001
Screech owl	Aroclor 1248	Diet	2 generations	ne	7.1	Eggshell thickness, egg production, hatching success, fledging success	McLane and Hughes 1980
Mallard	Aroclor 1242	Diet	12 weeks	105	nd	Eggshell thinning	Haseltine and Prouty 1980

nd – No data available

ne – No effect observed at any dose

Table A-5-16. Laboratory data for the effects of TEQs in eggs on birds

Test Organism	Analyte	Exposure route	Injection Time	LOAEL Egg TEQ Concentration (µg/kg ww)	NOAEL Egg TEQ Concentration (µg/kg ww)	Effect Endpoint	Reference
Ring-necked pheasant	TCDD	Egg albumin injection	No more than 5 days after eggs were obtained[a]	1	0.5	Embryo mortality	Nosek et al. 1993
Pigeon	TCDD	Egg air cell injection	Day 3.5 of 18 day incubation	1	nd	Hatchability	Janz and Bellward 1996
Great blue heron	TCDD	Egg air cell injection	Approximate midpoint of 28 day incubation	2	nd	Hatchability	Janz and Bellward 1996
Double-crested cormorant	TCDD	Egg yolk injection	Within 36-48 hrs of collection, prior to incubation[a]	4.0	1	Embryo mortality	Powell et al. 1997
Common tern	PCB 126[b]	Egg air cell injection	Day 4 of 18 day incubation	4.4	nd	Hatching success	Hoffman et al. 1998
American kestrel	PCB 77[c]	Egg air cell injection	Day 6 of 21 day incubation	5.0	nd	Hatching success	Hoffman et al. 1998
American kestrel	PCB 126[b]	Egg air cell injection	Day 6 of 21 day incubation	23	2.3	Hatching success	Hoffman et al. 1998
Double-crested cormorant	PCB 126[b]	Egg yolk injection	Within 36-48 hrs of collection, prior to incubation[a]	40	20	Embryo mortality	Powell et al. 1997

nd – No data available

PCB 126 TEF = 0.1; PCB 77 TEF=0.05

[a] No information available on length of incubation, or for pheasant, the age of the egg when obtained.

[b] The study used a TEF of 0.1 for PCB 126.

[c] The study used a TEF of 0.05 for PCB 77.

TEQ LOAEL for hatchability of 2 μg/kg ww (in eggs; Janz and Bellward 1996). A NOAEL (1 μg/kg ww) was available in Powell et al. (1997) for double-crested cormorant, a species in the same taxonomic order as heron. However, an injection resulting in an egg TEQ of 2 μg/kg was not tested in Powell et al. (1997), so it was not possible to compare the sensitivity of the two species at this dose. Because of potential effects on great blue heron at lower untested doses, the lowest available NOAEL and LOAEL (for ring-necked pheasant; 0.5 and 1 μg/kg ww in eggs) were chosen as the TEQ TRVs for birds.

A.5.2.3 TRVs for mammals

The COPCs for mammals were PCBs, arsenic, and lead.[93] This section discusses the general toxicity of each of these COPCs, presents toxicity data from the literature, compares toxicity studies to guidelines outlined in Section A.5.2, and chooses TRVs for otter and seal for each of their COPCs.

A.5.2.3.1 PCBs

PCBs have been reported to elicit a broad range of toxic effects in laboratory mammals under controlled exposure conditions, including lethality, hepatotoxicity, porphyria, body weight loss, dermal toxicity, thymic atrophy, immunosuppressive effects, reproductive and developmental effects, carcinogenesis, and neurotoxicity (for reviews of PCB toxicology, see Seegal 1996; Tilson et al. 1990; Safe 1984, 1990, 1991, 1992, 1994; Kimbrough 1985, 1987; Silberhorn et al. 1990; WHO 1993; Bolger 1993; Battershill 1994; Delzell et al. 1994). Review of the toxicology literature indicates that the potency of PCB mixtures depends on the chlorine content of the mixture and, in general, mixtures with higher chlorine content (i.e., Aroclors 1242, 1248, 1254, and 1260) are more toxic than mixtures with lower chlorine content (i.e., Aroclors 1221 and 1232). In general, the gastrointestinal tract of most mammals readily absorbs PCBs, but the absorption rate may be affected by the dose level and lipophilicity of the compound (Eisler 1986b; Van den Berg 1998). There is evidence for placental transfer of PCBs in mammals (Eisler 1986b), and PCBs can also accumulate in the lipid portion of milk, resulting in exposure to suckling animals.

Adverse reproductive effects (e.g., fertility, litter size, offspring survival) appear to be among the most sensitive *in vivo* endpoints of PCB toxicity in mammals (Golub et al. 1991; Rice and O'Keefe 1995; Hoffman et al. 1996). Reproductive success can be affected directly by toxic action on the differentiated reproductive tract or indirectly on systems that regulate reproduction (e.g., endocrine and central nervous systems). In laboratory studies, PCBs have been reported to elicit a broad range of direct and indirect effects that could conceivably lead to decreased reproductive function. For example, the liver appears to be one of the primary targets of PCB toxicity, and changes in the activity of liver enzymes can result in modulation of steroid hormone

[93] *COPCs for otter were PCBs, arsenic, and lead; the COPC for seal was PCBs (see Table A-5-1).*

levels, suggesting one mechanism by which PCBs could alter reproductive function. PCBs have also been implicated in the modulation of other systems important for reproduction, such as the central nervous system, adrenal gland, and thyroid hormone levels. Direct effects on the gonads and the female reproductive tract have been reported (Fuller and Hobson 1986). The precise mechanism by which PCBs cause reproductive effects in mammals remains unclear, but reproductive success appears to be a sensitive integrated endpoint of *in vivo* toxicity.

The most comprehensive studies of PCB toxicology in a wildlife mammalian species have been conducted with captive-bred mink. Mink also appears to be one of the most sensitive mammalian species tested (Fuller and Hobson 1986), and is therefore a good surrogate for assessment of risk to other mammals. Monkeys are also quite sensitive to PCBs, with reproductive effects generally observed at about 0.1 mg/kg bw/day (Barsotti et al. 1976; Allen et al. 1980; Truelove et al. 1982). However, effects levels from mink studies were used to develop TRVs for this assessment because of their greater taxonomic similarity to river otter, harbor seal, or other mammals that could use the LDW.

Table A-5-17 presents results of laboratory studies for reproductive and developmental endpoints for captive-bred mink. The methods and results of these studies were compared to the guidelines for TRV development discussed at the beginning of Section A.5.2, and the following observations were made:

- All studies were conducted during a critical life stage (reproduction), except the study by Hornshaw et al. (1986) that did not involve reproductive endpoints and was relatively short (4 weeks).
- Studies with Aroclor 1254 or Clophen A50 (similar in composition to Aroclor 1254) were most relevant to the LDW because of the mixtures tested in Table A-5-17; this mixture was most frequently detected in prey from the LDW. Studies by Aulerich and Ringer (1977), Aulerich et al. (1985), Hornshaw et al. (1986), and Kihlstrom et al. (1992) used Aroclor 1254 or Clophen A50.

Based on the above, the most appropriate study for TRV derivation with the lowest effect value was Wren et al. (1987), with a LOAEL of 0.15 mg/kg bw/day. The NOAEL was calculated as 0.015 mg/kg bw/day by applying an uncertainty factor of 10 to the LOAEL.

The discussion below provides additional qualitative information regarding risks from PCBs to mink, river otter, and harbor seal. However, these studies were not included in Table A-5-17 because they involved uncontrolled exposures to chemical mixtures or did not result in dietary effect thresholds.

Table A-5-17. Laboratory data for the effects of PCBs on mink

Test Organism	Analyte	Exposure Route	Exposure Duration	LOAEL[a] (mg/kg bw/d)	NOAEL[a] (mg/kg bw/d)	Effect Concentration (mg/kg ww)	Exposure Concentrations (mg/kg ww)	Effect Endpoint	Reference
Mink	Aroclor 1254	Diet	6 months	0.15	nd	1	0 and 1	Reduced growth rates of kits	Wren et al. 1987
Mink	Aroclor 1254	Diet	4 months	0.31	0.15	2	0, 1, 2, 5, and 15	Number of kits born alive	Aulerich and Ringer 1977
Mink	Aroclor 1254	Diet	88-102 days	0.38	nd	2.5	0 and 2.5	Number of kits whelped and born alive	Aulerich et al. 1985
Mink	Not reported	Diet	66 days	0.5	nd	0.5 mg/kg/d	0 and 0.5 mg/kg/d	Number of kits born alive	Jensen et al. 1977
Mink	Aroclor 1242	Diet	8 months	0.77	nd	5	0, 5, 10, 20, and 40	Reproductive failure	Bleavins et al. 1980
Mink	Aroclor 1254	Diet	4 weeks	1.5	nd	10	0, 10, 18, 32.4, 58.3, and 105	Weight gain in adults	Hornshaw et al. 1986
Mink	Aroclor 1254	Diet	3 months	1.64	nd	1.64 mg/kg/d	0 and 1.64 mg/kg/d	All whelps stillborn	Kihlstrom et al. 1992
Mink	Clophen A50	Diet	3 months	2.0	nd	2 mg/kg/d	0 and 2 mg/kg/d	All whelps stillborn	Kihlstrom et al. 1992
Mink	Aroclor 1016	Diet	8 months	2.7	nd	20	0 and 20	Birth weight and growth rate of kits, and adult mortality	Bleavins et al. 1980

nd –No data available

[a] Assuming a body weight of 0.975 kg (Aulerich and Ringer 1977) and ingestion rate of 0.150 kg/day ww (Bleavins et al. 1980)

Mink

A recent study by Restum et al. (1998) examined multigenerational reproductive success of mink fed field-collected fish contaminated with a number of organic pollutants, including PCBs, dioxins, furans, and pesticides. In this study, seven-month-old mink were fed diets containing various amounts of contaminated carp from Saginaw Bay, Lake Huron. A diet of fish containing 0.004 mg PCB/kg bw/day was correlated with a reduced growth rate of kits in the F1 generation. However, this study does not support estimation of a PCB effect threshold because of the presence of multiple contaminants in the carp. It only has quantitative relevance to mink exposed to chemical mixtures similar to those found in the Saginaw Bay fish. Furthermore, the presence of other, uncharacterized, toxic chemicals in the Saginaw Bay fish was suggested by results of a dioxin equivalency toxicity test performed by the authors, which suggests the presence of Ah-R agonists (and therefore dioxin-like biological activity) in addition to the detected chlorinated organic compounds (Tillitt et al. 1996).

River Otter

The reproductive effects of PCBs in river otter have not been extensively investigated in controlled laboratory studies, presumably because of the difficulties in rearing this species in captivity. For purposes of this assessment, it was conservatively assumed that river otter are as sensitive to PCBs as mink, a related mustelid species. However, the limited available data indicate river otter should be less sensitive to PCBs than mink.

Davis (1993) fed wild-caught river otter diets containing different amounts of PCB contaminated carp (average total PCB concentration of 5.7 mg/kg ww) from Saginaw Bay, Michigan, and monitored food consumption, body weight, PCB concentrations in tissue, and several blood and histopathological endpoints. The carp contained multiple chemicals, thus preventing derivation of a PCB effect threshold. Of the chemical mixture, the dietary PCB concentrations fed to the otter were 0.03, 1.90, 3.67, and 5.22 mg/kg ww, a range that brackets the dietary threshold for mink of 2 mg/kg ww (Aulerich and Ringer 1977). During the first two weeks of the trial, food consumption and body weights were markedly reduced. Moreover, convulsions, loss of coordination, paralysis, and loss of consciousness were observed in river otter fed a diet of 60% carp. Similar but less severe effects were observed in river otter fed less carp. The authors identified these symptoms as being consistent with symptoms of thiamine deficiency observed in other mammals, and the adverse effects initially observed were reversed following supplementation of the diet with thiamine. Based on these results, the authors concluded the observed adverse effects were attributable to a thiamine deficiency rather than PCB toxicity. Moreover, the authors concluded that "the otter may not be as sensitive to PCBs and perhaps other organochlorine contaminants as mink," because "although the otter had tissue residue concentrations of PCBs similar to those observed in other species fed comparable concentrations of

PCBs for similar exposure periods, the otter did not exhibit the typical clinical signs usually associated with PCB toxicity."

Field studies on mink and river otter populations along the lower Columbia River appear to support the hypothesis that river otter are no more sensitive than mink to the effects of PCBs. PCB concentrations in river otter liver from this area are reported to range from 4.8 to 23 mg/kg liver ww (Henny et al. 1981). This range was somewhat higher than that measured in livers of captive mink that died or failed to produce young after chronic dietary exposure to PCBs (i.e., mean range of 0.87 to 11.99 mg/kg liver ww [Platonow and Karstad 1973]). Columbia River otter populations are considered stable despite such PCB exposure (Columbia Basin Fish and Wildlife Authority 1996; Tetra Tech 1996). In fact, the Columbia River has the highest published estimate of river otter density in North America (Tetra Tech 1996). Collectively, these data suggest healthy river otter populations can be maintained at PCB exposure concentrations that are toxic to mink in a laboratory setting.

Populations of European otter (*Lutra lutra*), a species related to the American otter (*Lutra canadensis*), have declined widely in Europe during the last 30 years. PCBs have been widely implicated as a potential cause of this decline (Mason et al. 1992; Mason and O'Sullivan 1992; Mason 1993; Mason and Madsen 1993; Mason and MacDonald 1993; Mason and MacDonald 1994; Tans et al. 1996; Sjöåsen et al. 1997; Gutleb and Kranz 1998; Mason 1998). The suspected role of PCBs in the decline of European otter was not based on controlled laboratory experiments, but rather on relationships between population estimates and empirical data on PCB concentrations in tissue of wild animals and scat, including data collected by Jensen et al. (1977). Critical PCB concentrations associated with the onset of reproductive adverse effects for river otter have been suggested to be 30 to 50 mg/kg in fat, or 16 mg/kg in scat (De Vries 1989; Mason 1989, as cited by Mason and Madsen 1993). Populations of European otter that are in decline are reported to have mean PCB concentrations greater than 50 mg/kg lipid and those that have remained stable or are increasing have mean PCB concentrations lower than 50 mg/kg lipid. However, these apparent thresholds have not been tested with controlled exposures.

Qualitatively, the available data suggest that river otter should not be more sensitive to PCB exposure than mink, and that use of mink toxicity data to assess risk to river otter should be protective.

Harbor Seal

There have been no controlled feeding studies exposing harbor seals to PCBs. Investigators who study wild populations have suggested a link between mass mortalities in harbor seals and immunotoxicity from chlorinated organic contaminants, including pesticides, PCBs, polychlorinated dibenzodioxins (PCDDs), and polychlorinated dibenzofurans (PCDFs) (Ross et al. 1996). High, acute doses of chlorinated compounds, including PCBs, have long been reported to adversely affect immune responses and increase susceptibility to disease in laboratory animals (Vos

1977), though the relevance of this to environmentally realistic exposure levels and pathways in wildlife species remains unknown. Van Loveren et al. (2000) fed captive harbor seals two diets consisting of field-collected fish, one that contained high levels of chlorinated organics, including PCBs, pesticides, dioxins and furans, and one that contained low levels of these compounds. Although no integrated endpoint of immune function was measured (e.g., ability to resist infection), seals fed fish with higher contaminant concentrations showed suppression of seven biochemical measures of immune function relative to those fed the low-contaminant diet. Because the food source was contaminated with a mixture of compounds, the results cannot be used to derive a PCB effect threshold. It is also unclear whether the biomarkers of exposure reported are indicative of adverse effects at the individual or population level, but such studies have raised concerns about immunological effects of chlorinated organics on marine mammals. Immunological effects of PCB exposure may be important in mammals, but data to estimate an adverse effect threshold for immunosuppression are not yet available.

A.5.2.3.2 Arsenic

Mammalian effects from chronic exposure to inorganic arsenic may include weakness, paralysis, conjunctivitis, dermatitis, decreased growth, liver damage, and developmental effects in offspring (Eisler 1988b). Early developmental stages are most sensitive to arsenic exposure.

Table A-5-18 summarizes available information from the only two studies identified in the literature related to reproductive and developmental toxicity, which are considered the most sensitive endpoints. No studies were found that examined the effects of arsenic on wildlife species, and studies using relevant exposure scenarios and effects endpoints were limited. In addition, no relevant chronic studies were found with growth or survival endpoints. Of the two studies presented, the LOAEL from Schroeder and Mitchener (1971) was most appropriate for the arsenic TRV because the study by Nemec et al. (1998) used gavage as an exposure route and was conducted for a shorter time period. However, it should be noted that this LOAEL (1.26 mg/kg bw/day) was based on exposure to arsenic in drinking water and food, and therefore may overestimate dietary risk because gastrointestinal absorption may be higher for chemicals ingested via drinking water (Sample et al. 1996). A NOAEL value was not available, so it was estimated at one tenth the LOAEL, or 0.126 mg/kg bw/day.

Table A-5-18. Laboratory data for the effects of arsenic on mammals

Test Organism	Analyte	Exposure Duration	Exposure route	LOAEL (mg/kg bw/d)	NOAEL (mg/kg bw/d)	Effect Concentration (mg/kg ww or mg/L)	Exposure Concentrations (mg/kg ww or mg/L)	Effect Endpoint	Reference
Mouse	Soluble arsenite	3 generations	Drinking water and diet	1.26[a]	nd	5 mg/L in water and 0.06 mg/kg ww in food	5 mg/L in water and 0.06 mg/kg ww in food	Decreased litter size	Schroeder and Mitchener 1971
New Zealand white rabbit	Arsenic acid	13 gestational days	Gavage	3.0	0.75	3.0 mg/kg/d ww	0, 0.19, 0.75, and 3.0 mg/kg/d ww	Maternal mortality and body weight, fetal mortality	Nemec et al. 1998
Swiss albino mouse	Arsenic acid	10 gestational days	Gavage	24	7.5	24 mg/kg/d ww	0, 7.5, 24, and 48 mg/kg/d ww	Maternal mortality, fetal weight, developmental effects	Nemec et al. 1998

nd – No data available

[a] Assuming a body weight of 0.03 kg, food ingestion rate of 0.0055 kg/d ww, and water ingestion rate of 0.0075 L/day (Sample et al. 1996)

A.5.2.3.3 Lead

Lead has been associated with neurotoxicity, muscular paralysis, inhibition of heme synthesis, kidney and liver damage, and reproductive impairment in mammals (Eisler 1988a). It is also a suspected carcinogen. Reproductive endpoints are the most sensitive endpoints. Table A-5-19 presents results of laboratory studies considered in lead TRV development. These studies were compared to guidelines for TRV development discussed in Section A.5.2, as follows:

- All studies were conducted over 12 weeks or with reproductive endpoints
- None of the test species were closely related to otter or seal
- All studies used inorganic forms of lead, except Odenbro and Kihlstrom (1977), which used triethyl lead
- Diet is the most relevant pathway because of potentially higher absorption from drinking water (Sample et al. 1996); all studies involved drinking water alone or in combination with food, except for Azar et al. (1973), who used food alone

Based on the comparison to guidelines, all studies were similar with the exception of Azar et al. (1973), which used food exposure only, and Odenbro and Kihlstrom (1977), which used an organic form of lead. Organolead compounds are generally more toxic than inorganic forms, but most lead accumulation and toxicity in animals results from inorganic lead, due to its widespread use and distribution in the environment (Hoffman et al. 1995). Data were not available for organolead compounds in LDW fish. Because the organolead study had the lowest effect value of all studies, and because of the uncertainty in exposure of LDW fish to organolead, the NOAEL and LOAEL from the Odenbro and Kihlstrom (1977) study were used to provide more conservative values for the purpose of this evaluation. This study also used a water exposure route, which could result in higher absorption than diet, thus providing a more conservative approach. The resulting NOAEL and LOAEL values used for TRVs were 0.5 and 1.5 mg/kg bw/day, respectively (Odenbro and Kihlstrom 1977).

Table A-5-19. Laboratory data for the effects of lead on mammals

Test Organism	Analyte	Exposure Duration	Exposure route	LOAEL (mg/kg bw/d)	NOAEL (mg/kg bw/d)	Effect concentration (mg/kg ww or mg/L)	Exposure concentrations (mg/kg ww or mg/L)	Effect Endpoint	Reference
Mouse	Triethyl lead chloride	Day 3-5 after mating	Oral in water by gavage	1.5	0.5	1.5 mg/kgbw/d	0, 0.5, and 1.5 mg/kg bw/d	Decreased frequency of implanted ova	Odenbro and Kihlstrom 1977
Rat	Soluble lead	3 generations	Drinking water and diet	3.3[a]	nd	25 mg/L in water and 0.20 mg/kg ww in food	25 mg/L in water and 0.20 mg/kg ww in food	Offspring mortality and runts	Schroeder and Mitchener 1971
Mouse	Soluble lead	3 generations	Drinking water and diet	6.3[b]	nd	25 mg/L in water and 0.20 mg/kg ww in food	25 mg/L in water and 0.20 mg/kg ww in food	Offspring mortality, runts, discontinuation of breeding	Schroeder and Mitchener 1971
Rat	Lead acetate	12 weeks	Drinking water	8.9[c]	4.7[c]	50	0.5, 5, 25, 50, and 250	Growth of females through pregnancy and lactation	Kimmel et al. 1980
Rat	Lead acetate	2 years	Diet	80[d]	8.0[d]	1000	10, 50, 100, 1,000, and 2,000	Offspring weight and kidney damage	Azar et al. 1973
Albino mouse	Lead acetate	11 weeks	Drinking water	200[e]	nd	800	0.08%	Pup weight and survival	Sharma and Kanwar 1985

nd – No data available

[a] Assuming a body weight of 0.35 kg, food ingestion rate of 0.028 kg/d ww, and water ingestion rate of 0.046 L/day (Sample et al. 1996).

[b] Assuming a body weight of 0.03 kg, food ingestion rate of 0.0055 kg/d ww, and water ingestion rate of 0.0075 L/day (Sample et al. 1996).

[c] Median dose was calculated in the study with body weights and food ingestion rates that were not presented.

[d] Assuming a body weight of 0.35 kg and food ingestion rate of 0.028 kg/d ww (Sample et al. 1996).

[e] Assuming a body weight of 0.03 kg and water ingestion rate of 0.0075 L/day (Sample et al. 1996).

A.5.3 SUMMARY OF WILDLIFE ASSESSMENT

A.5.3.1 Exposure assessment

The exposure assessment provided an estimate of each ROC's exposure to COPCs through ingestion of prey and incidental sediment ingestion. Exposure doses were calculated for each ROC/COPC pair, and expressed as mg COPC ingested per kg body weight per day. Estimates of dietary composition and site usage were made using site-specific information, if available, along with species life history information. Exposure dose calculations were made using SWA sediment concentrations and the lower of either the maximum or 95% UCL mean prey tissue concentrations. A summary of exposure doses for wildlife is presented in Table A-5-20. In addition, concentrations of PCBs and TEQs in great blue heron eggs are presented.

A.5.3.2 Effects assessment

The effects assessment established doses representing dietary thresholds of effects for each ROC/COPC pair. Threshold concentrations of PCBs and TEQs in bird eggs were also established. The toxicity literature was searched and relevant data for birds and mammals were compiled and screened against a set of guidelines to select the most appropriate TRVs. TRVs for both no-effects and low-effects data were chosen, as summarized in Tables A-5-21 and A-5-22.

Table A-5-20. Dietary exposure doses and egg concentrations for ROC/COPC pairs identified in Table A-5-1

ROC	Dietary Exposure (mg/kg bw/d ww)							Egg Concentration (mg/kg egg ww)	
	PCBs	BEHP	As	Cu	Hg	Pb	Zn	PCBs	TEQs
Sandpiper	0.363	0.419	ne	27.5	ne	8.23	23.9	ne	ne
Heron	0.109	ne	ne	ne	0.0156	0.0825	ne	47	$1.3x10^{-3}$
Eagle	0.0286–0.114	ne	ne	ne	0.0026–0.0103	0.0104-0.0415	ne	ne	ne
Otter	0.128	ne	0.774	ne	ne	0.0619	ne	ne	ne
Seal	0.0099	ne	ne	ne	ne	ne	ne	ne	ne

ne – Not evaluated in the EEA (dietary exposure screened out in the problem formulation or egg data not available)

Table A-5-21. Dietary TRVs for ROC/COPC pairs (mg/kg bw/day ww)

ROC	PCBs		BEHP		As		Cu		Hg		Pb		Zn	
	NOAEL	LOAEL	NOAEL	LOAEL	NOAEL	LOAEL	NOAEL	LOAEL	NOAEL	LOAEL	NOAEL	LOAEL	NOAEL	LOAEL
Sandpiper	0.41	0.94	5.1	350	ne	ne	47	62	ne	ne	2.0	20	82	123
Heron	0.41	0.94	ne	ne	ne	ne	ne	ne	0.0091	0.091	2.0	20	ne	ne
Eagle	0.41	0.94	ne	ne	ne	ne	ne	ne	0.0091	0.091	2.0	20	ne	ne
Otter	0.015	0.15	ne	ne	0.126	1.26	ne	ne	ne	ne	0.5	1.5	ne	ne
Seal	0.015	0.15	ne	ne	ne	ne	ne	ne	ne	ne	ne	ne	ne	ne

ne – Not evaluated in the EEA (screened out in the problem formulation)

Table A-5-22. Egg TRVs for heron (mg/kg egg ww)

	PCBs		TEQs	
	NOEC	LOEC	NOEC	LOEC
Heron	7.1	16	0.5×10^{-3}	1.0×10^{-3}

TEQ – Summation of toxicity equivalence factors (TEF)s multiplied by the corresponding concentration of PCB congeners

A.6 Exposure and Effects Assessment: Plants

Emergent rooted aquatic plants were selected as a ROC in the problem formulation. For plants, four COPCs (lead, mercury, PCBs, and zinc) were identified. These COPCs were identified by comparing maximum chemical concentrations in intertidal and marsh sediments to toxicity benchmarks available for rooted terrestrial plants (Efroymson et al. 1997) and to background concentrations. Background concentrations were based on Puget Sound reference sediments and performance standards (PTI 1991) and soils (Ecology 1994).

This section assesses potential exposure of rooted aquatic plants in the LDW to these COPCs. In addition, a detailed evaluation of the available effects data is presented. Uncertainties inherent in the use of terrestrial plants as a surrogate for rooted aquatic plants, and the use of marsh sediment data, are discussed in the uncertainty assessment (Section A.7.4.2).

A.6.1 Exposure Assessment

Exposure of sediment-associated chemicals to rooted aquatic plants was assessed through the evaluation of COPC concentrations in sediments in potential macrophytic habitat. The dominant plant species in the LDW and their habitat preferences are:

- *Carex* species (sedges) can be found at edges of estuarine meadows, marshes, tidal flats, sandy beaches, swamps, gravelly shores, bars, banks, cutbanks, grassy slopes, seeps, roadsides, heaths, rocky runnels, ditches, clearings, and disturbed areas.
- *Scirpus* species (bulrushes) usually occur in marshes, shores, shallow water (fresh and brackish), swamps, sloughs, stream banks, wet ditches and meadows, bogs, and fens.
- *Salicornia* species (grassworts) are common in salt marshes and on tideflats and beaches. They are generally absent in areas with strong wave action and surf.
- *Distichlis* species (salt grass) grow in tidal marshes and along shorelines.
- *Atriplex* species (salt bush) are found in tidal and salt marshes, saline soils, tideflats, and along beaches.

The quality of estuarine macrophytic habitat is generally governed by salinity and percent tidal immersion. In the LDW, there are currently a total of 1.7 hectares of suitable habitat for macrophytes, primarily limited to portions of Kellogg Island and other small intertidal areas with vegetated habitat (USFWS 2000; Map A-6-1, Attachment A.1). Thus, sediment concentrations (both maximum and 95% UCL of the mean) of the four COPCs identified in the problem formulation are presented for both intertidal sediments and marsh areas (where rooted plants are more likely to be

present) (Table A-6-1). Concentration ranges and 95% UCL mean concentrations of all four COPCs were lower in marsh areas than in intertidal sediment areas as a whole, although far fewer sediment samples were available in these areas for comparison (n = 7 in marsh areas vs. n = 448 in intertidal areas). Thus, plants are likely to have lower exposure to sediment-associated chemicals in their most suitable habitats.

Table A-6-1. Comparison of COPC concentrations in marsh and intertidal habitats of the LDW to Puget Sound background concentrations

Chemical	Range (and 95% UCL on the mean)[a] Sediment Concentration in Intertidal (mg/kg dw)	Range (and 95% UCL on the mean)[a] Sediment Concentration in Marshes (mg/kg dw)[b]	Background Soil and Sediment Concentration Range (mg/kg dw)
Lead	2.0–23,000 (410)	9.3–330 (160)	0.1U–24[c]; 29.6[d]; 20[e]
Mercury	0.020–4.6 (0.23)	0.090–0.37 (0.25)	0.01—0.28[c]; 0.0944[d]; 0.15[e]
PCBs (total-calculated)	0.00030– 223 (3.0)	0.020–9.4 (1.7)	0.0031–0.050U[c]; 0.047[e]
Zinc	16–6,400 (312)	56–160 (133)	15–101J[c]; 132.5[d]; 103[e]

U - Undetected

J - Estimated

[a] Nondetects were treated as half the detection limit in the 95% UCL calculations

[b] Maximum concentrations of COPC within 50 m of marsh habitat (per USFWS designation) (n=7 stations: DR013, DR014, DR061, DR263, DR264, DR270, DR271; see RI Maps 2-5a through 2-5k)

[c] PTI (1991) (range of concentrations from Puget Sound sediment reference areas)

[d] Ecology (1994) (maximum concentration in Puget Sound soil reference areas)

[e] PTI (1991) (proposed reference area performance standard [i.e., sites with concentrations lower than these standards are suitable for reference area classification])

Lead, mercury, zinc, and PCBs 95% UCL mean and maximum concentrations in marsh areas were greater than background concentrations (except for 95% UCL of the mean concentration of mercury and zinc in marsh areas, which were similar to background concentrations). In the risk characterization (Section A.7.4), exposure of plants to these four COPCs is addressed using the range of sediment concentrations in marsh areas. Potential exposure in intertidal areas is discussed in Section A.7.4.2, the uncertainty assessment.

A.6.2 Effects Assessment

In this section, available toxicology literature regarding growth and survival endpoints for rooted plants are presented for lead, mercury, zinc, and PCBs. The Oak Ridge National Laboratory guidance document for plants (Efroymson et al. 1997) was reviewed, as well as citations identified through a search of the Science Citation index. Based on this review, no studies were found that assessed toxicity of these COPCs in contaminated sediments to halophytic plants like the grasses, sedges, and rushes found in the LDW (*Carex, Scirpus, Salicornia, Distichlis,* and *Atriplex*). Thus, this section summarizes the available terrestrial rooted plant literature (i.e., chemical concentrations in soils associated with adverse effects). To be acceptable, it was

necessary that studies included synoptic measurements of COPC concentrations in soil and observed effects and adequate controls.

A summary of relevant toxicity studies is presented in Table A-6-2; ten studies were identified for lead, five studies for zinc, and three studies were identified for PCBs. One study was identified for mercury (Panda et al. 1992); however, it was deemed unacceptable because it was a field study with co-occurring contaminants. Other studies that were reviewed but not used had:

- measurement of exchangeable zinc in soil rather than total zinc (Gall and Barnette 1940)
- high variability in control and exposure tests (Carlson and Bazzaz 1977)
- lack of contaminant concentrations in soil (just soil solution concentration was reported) (Davis et al. 1978)
- the presence of co-occurring contaminants (Panda et al. 1992)

Table A-6-2. Summary of plant toxicity studies and soil-based NOECs and LOECs

Citation	Chemical	Plant Species	Duration	Effect	NOEC (mg/kg dw)[a]	LOEC (mg/kg dw)[a]
Dixon 1988	Pb ($PbCl_2$)	red oak seedlings [b]	16 weeks	reduction in weight and leaf area	9	21
Rolfe and Bazzaz 1975	Pb ($PbCl_2$)	autumn olive seedlings	4 or 7 weeks[c]	reduction in transpiration and photosynthesis	160	320
Miles and Parker 1979	Pb ($PbCl_2$)	little bluestem and black-eyed susan (from seed)	12 weeks	reduction in root and shoot weight	na	177 [d]
Hassett et al. 1976	Pb ($PbCl_2$)	corn (from seed)	7 days	reduction in root length	100	250
Miller et al. 1977	Pb	corn (from seed)	17 days	reduction in plant weight		125
Khan and Frankland 1984	Pb ($PbCl_2$ [oat], $PbCl_2$:PbO [radish], $PbCO_3$ [wheat]	oat, radish, wheat seedlings	42 days	reduction in root weight	100 (oat), 500 (radish)	500 (oat), 1000 (radish), 1000 (wheat)
Khan and Frankland 1983	Pb ($PbCl_2$, PbO)	radish (from seed)	42 days	reduction in root growth	100 ($PbCl_2$)	500 ($PbCl_2$), 1,000 (PbO)
Khan and Frankland 1983	Pb ($PbCl_2$, PbO)	radish (from seed)	42 days	reduction in shoot growth	1,000 ($PbCl_2$) 5,000 (PbO)	5,000 ($PbCl_2$), 10,000 (PbO)
Muramoto et al. 1990	Pb (PbO)	wheat (from seed)	23 weeks	reduction in root weight		30,000
John and van Laerhoven 1972	Pb ($PbCl_2$, $PbCO_3$, $Pb(NO_3)_2$)	oat and lettuce seedlings	21; 30 days	reduction in plant weight	1,000 (oat) [d]	1,000 (lettuce) [d]
Carlson and Rolfe 1979	Pb ($PbCl_2$)	rye grass and fescue (from seed)	30 days	reduction in clipping weight	1,000 (in fertilized soil)	5000
Strek and Weber 1980	PCBs (Aroclor 1254)	fescue, sorghum, corn, soybean, beets, pigweed	seed to 16 days	reduction in height and/or fresh weight	20 (pigweed); 1,000 (corn, sorghum, fescue)	40 (pigweed); 1,000 (soybeans, beets)
Strek and Weber 1982	PCBs (Aroclor 1254)	pigweed	28 days	reduction in plant height (23%)	50	100
Weber and Mrozek 1979	PCBs (Aroclor 1254)	soybean	26 days	reduction in shoot weight (27%)	10	100

Citation	Chemical	Plant Species	Duration	Effect	NOEC (mg/kg dw)[a]	LOEC (mg/kg dw)[a]
Hagemeyer et al. 1993	Zn ($ZnSO_4$)	2-yr-old beech tree	2 seasons	reduction in annual growth ring and apical shoot length	15	66
Aery and Sakar 1991	Zn ($ZnSO_4$)	soybean (from seed)	seed to flowering	reduction in seed number, nodule weight, and seed weight [e]	10 (seed #), 100 (nodule wt), 2,500 (seed #)	25 (seed #), 500 (nodule wt), 5,000 (seed #)
Lata and Veer 1990	Zn	spinach and coriander (from seed)	60 days	reduction in shoot weight		49 [d, f]
White et al. 1979	Zn ($ZnSO_4$)	soybean	4 weeks (pH .5) [g]	reduction in leaf weight	262	327
Muramoto et al. 1990	Zn (ZnO)	wheat and rice (from seed)	23 weeks (wheat), 15 weeks (rice)	reduction in wheat grain yield and growth	[h]	10,000

na – Not available

[a] Concentration in soil

[b] Seedlings were inoculated with ectomycorrhizae, which naturally occurs with red oak

[c] Paper is unclear

[d] Only one concentration was tested

[e] Concentrations of 10-25 mg/kg Zn promoted soybean growth

[f] Concentration in soil converted assuming density of soil is similar to that of quartz (2.65 g/mL)

[g] Effects in soil at pH 5.5 were also evaluated, but pH 6.5 results were deemed more relevant (approximate LDW sediment pH is 7.5)

[h] Significant effects were not reported for rice or wheat at 1,000 and 3,000 mg/kg ZnO in soils, but paper is too unclear to determine if statistical evaluations were conducted

The ranges of NOECs and LOECs reported for lead, mercury, PCBs, and zinc are summarized in Table A-6-3.

Table A-6-3. Summary of soil NOEC and LOEC (mg/kg dw) ranges for plants

CHEMICAL	NOECs	LOECs
Lead	9–5,000 (n = 9)	21–30,000 (n = 10)
Mercury	na	na
PCBs	10–1,000 (n = 3)	40–1,000 (n = 3)
Zinc	10–2,500 (n = 4)	25–5,000 (n = 5)

na – Not available

Of the studies summarized in Tables A-6-2, none of the test species were directly related to species in the LDW, and none of the studies were conducted under estuarine marsh conditions. In addition, large and overlapping ranges of NOECs and LOECs were observed for lead, PCBs, and zinc. Only one study was identified for mercury (Panda et al. 1992), but the results of this study were not relevant due to the presence of co-occurring chemicals (discussed below). The uncertainties associated with the effect data for plants are discussed further in the uncertainty assessment (Section A.7.4.2).

The lowest NOEC (9 mg/kg) and LOEC (21 mg/kg) for lead were determined from toxicity tests with red oak seedlings (Dixon 1988). These concentrations are lower than regional background sediment lead concentrations (Table A-6-1). The next lowest LOEC, 125 mg/kg, was based on reduction in corn weight (Miller et al. 1977c), and the next lowest NOEC, 100 mg/kg, was reported for corn, oats, and radishes (Table A-6-2). The selected NOEC and LOEC for lead were 100 and 125 mg/kg, respectively. These concentrations were selected because the LOEC is the lowest LOEC that is higher than background sediment lead concentrations, and the NOEC was the highest NOEC less than the selected LOEC.

For mercury, only one study was identified relating concentrations of mercury in soil to adverse effects on vascular plants. Panda et al. (1992) reported a NOEC of 35 mg/kg and a LOEC of 64 mg/kg associated with reduced plant height and seed germination in barley seeds. However, the soil matrix in this study was solid waste deposits from a chloralkali plant, so results were not relevant to plants in the LDW. In the Efroymson et al. (1997) plant benchmark compilation, Kabata-Pendias and Pendias (1984) was cited as reporting that unspecified toxic effects on plants grown in surface soil were observed at 0.3 mg/kg mercury. However, review of this study indicated that Kabata-Pendias and Pendias (1984) provided an overview of two papers (Shacklette et al. 1978 and Davis et al. 1978). Shacklette et al. (1978) reported observed mercury concentrations in plants, but no toxicity data. Davis et al. (1978) reported toxic effects (reduction of plant yield of dry matter) on spring barley at 4 mg/L mercury in solution. This solution concentration resulted in a plant tissue concentration of 3 mg/kg dw. Based on information provided in these papers, it is not clear how the

0.3 mg/kg in soil threshold cited in Efroymson et al. (1997) was derived, and thus it was not used in this assessment. Thus, NOEC or LOEC TRVs for mercury were not available for use in this assessment; the risk characterization (Section A.7.4.1) will instead discuss the exposure concentrations of mercury in the marsh areas of the LDW relative to background concentrations and uncertainties regarding toxicity data for mercury will be acknowledged in the risk characterization and uncertainty assessment (Section A.7.4).

Three studies were identified to assess the potential impact of PCB exposures on plants. NOECs ranged from 10 to 1,000 mg/kg in soil for five different plants, and LOECs ranged from 40 to 1,000 mg/kg. LOECs for pigweed, the most sensitive plant tested, varied from 40 to 100 mg/kg (Strek and Weber 1980, 1982), and thus 40 mg/kg was selected as the plant LOEC for PCBs. A NOEC of 20 mg/kg was selected because it was the highest NOEC below the LOEC of 40 mg/kg for pigweed available. Note that there is uncertainty in the NOEC and LOEC, because Strek and Weber (1982) report a NOEC of 50 mg/kg for pigweed and plant height.

For zinc, half of the NOECs and LOECs presented in Table A-6-2, as well as the Efroymson et al. (1997) screening benchmark (50 mg/kg), were below background zinc soil or sediment concentrations (15 - 130 mg/kg; Table A-6-1). LOECs ranged from 25 to 5,000 mg/kg and NOECs ranged from 10 to 2,500 mg/kg. The lowest LOEC that was also greater than regional background sediment and soil concentrations was selected (327 mg/kg associated with a reduction in leaf weight; White et al. 1979). For consistency, the NOEC from this same study was selected as well (262 mg/kg).

A.6.3 Summary of Rooted Plants Assessment

A.6.3.1 Exposure assessment

Sediment concentrations of the four COPCs identified in the problem formulation for rooted plants were compiled for marsh and intertidal areas in the LDW, and compared to regional soil and sediment background concentrations. In marsh areas, where plants are most likely to grow based on habitat constraints, lead, mercury, zinc, and PCBs had maximum concentrations that exceeded background concentrations (Table A-6-1). The concentration range of these four COPCs in the marsh areas will be used in the risk characterization.

A.6.3.2 Effects assessment

No toxicity data were available for estuarine rooted plants. Thus, toxicity data for terrestrial rooted plants in soils were used. Large and overlapping ranges of NOECs and LOECs were observed for lead, PCBs, and zinc. Only one study was identified for mercury, but it was not acceptable due to the presence of co-occurring contaminants. NOECs and LOECs selected for the COPCs are presented in Table A-6-4. Application of this toxicity information is highly uncertain because the relative sensitivity of the species tested and those present in the marsh areas of the LDW is unknown; the

exposure conditions employed in the toxicity study and in the LDW are different (soils vs. sediments); and the ranges of NOECs and LOECs were generally large and overlapping.

Table A-6-4. NOECs and LOECs selected to assess risks to rooted plants

COPC	NOEC mg/kg dw soil	LOEC mg/kg dw soil
Lead	100	125
Mercury	na	na
Zinc	262	327
PCBs	20	40

na – Not available

A.7 Risk Characterization and Uncertainty Assessment

This section presents the risk characterization for each ROC/COPC pair determined in the problem formulation (Section 2) and discussed in the exposure and effects assessments of this Phase 1 ERA. The risk characterization section for each receptor group (benthic invertebrates, fish, wildlife, and plants) consists of a risk estimation, an uncertainty assessment, and a risk conclusion section. The risk estimation presents the HQs[94] calculated for each ROC/COPC pair (i.e., it synthesizes the exposure and effects data used in this Phase 1 ERA). Uncertainties inherent in these HQs and in the Phase 1 ERA problem formulation and exposure and effects assessment approach are discussed in the uncertainty assessment. Problem formulation uncertainties are focused on selection of ROCs, assessment endpoints, and exposure pathways. The exposure assessment discussion highlights uncertainties related to either the availability or relevance of site-specific data to estimate or measure exposure, as well as any parameters used in modeling exposure. The effects assessment discussion highlights uncertainties in the availability and relevance of toxicological data, the majority of which were selected from the literature. The results of the HQ calculations and the uncertainty assessment are then integrated in the risk conclusions.

The magnitude of the preliminary risk estimate and the uncertainty in this estimate will be used in the data gaps process to determine which uncertainties are most critical to address in Phase 2. In ERAs, HQs greater than 1 are generally regarded as indicating a potential for adverse effects, particularly if the HQ is based on an effects concentration (or dose) at which adverse effects were observed. These HQs are referred to as lowest observed effects concentration (LOEC)- or lowest observed adverse effects levels (LOAEL; for doses)-based HQs. In other words, exposure is believed to be sufficiently high that adverse effects are more likely to occur. HQs are also calculated based on a no observed effects concentration (NOEC) or no observed

[94] HQ = exposure concentration (or dose)/ concentrations (or dose) associated with adverse effects

adverse effects level (NOAEL), for doses. Note that although a NOEC-based HQ may exceed 1, the potential for adverse effects is uncertain because the true threshold for effects occurs at a concentration somewhere between the NOEC and LOEC. Therefore, it is important to calculate both types of HQs to estimate the potential for adverse effects.

The Phase 1 ERA identifies sediment-associated COPCs that may be of concern to ecological species. However, because this Phase 1 ERA was based on a relatively small tissue data set and used highly conservative assumptions, not all chemicals identified as chemicals of concern will be risk drivers for the site at the conclusion of Phase 2. On the other hand, chemicals now believed to pose low risk based on the existing data set may be found to pose a higher risk once a more comprehensive dataset has been gathered in Phase 2. Thus, in the problem formulation of the Phase 2 ERA, any additional data gathered to fill data gaps, or identified through other means, will be used to determine the COPCs for Phase 2. The entire ERA COPC selection process is presented in Figure A-7-1 for clarity.

To aid in identifying specific data gaps for further analysis, tables in this section summarize key topics of uncertainty and qualitatively rank these topics regarding their level and direction of uncertainty, their potential to impact risk conclusions, and the feasibility of reducing the uncertainty either through literature-based analysis, field studies, or additional investigations. Based on this analysis and further discussion with stakeholders, the data gaps memorandum will recommend additional field work to address critical data gaps. In addition to the more detailed analyses in the Phase 2 risk assessments as well as feasibility considerations, these additional data will enable informed decisions by the agencies regarding any potential sediment cleanup actions in the LDW.

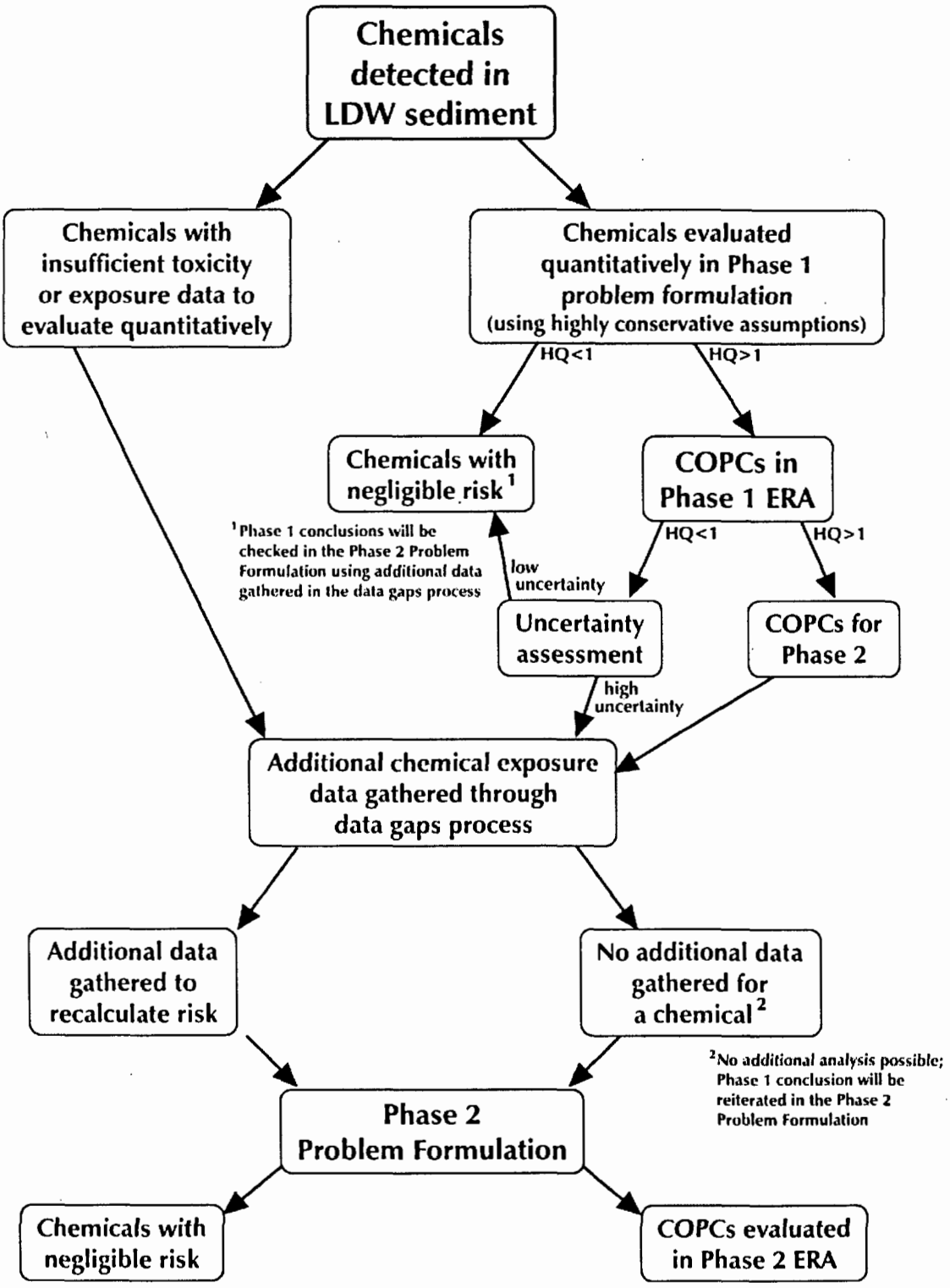

Figure A-7-1. Phased process by which COPCs will be addressed

A.7.1 Risk Characterization for Benthic Invertebrates

This section characterizes risks to benthic invertebrates closely associated with sediment, such as amphipods and polychaetes, and more mobile, higher-trophic level invertebrates, such as Dungeness crab, that may travel over relatively greater distances than other invertebrates.

The risk characterization for infaunal and epibenthic invertebrates was based primarily on a prediction of effects through the comparison of available surface sediment chemistry data with available sediment quality standards and guidelines. The large volume of surface sediment chemistry data presented in the exposure assessment (Section A.3.1) represents a reasonably thorough portrayal of the chemical exposure regime for most COPCs identified in the problem formulation. However, site-specific measures of direct effects for sediment-associated benthic invertebrates (e.g., toxicity tests, benthic community assessment) were far fewer. Only 10 sediment samples were tested for toxicity (as measured by biological standards specified in the SMS). The limited existing benthic macroinvertebrate community data cannot be evaluated relative to the SMS because a reference site sample was not collected for comparison. Additional discussion of these data is provided in the uncertainty assessment (Section A.7.1.2). Accordingly, the risk characterization relied on the prediction of effects, using available sediment quality standards and guidelines, rather than on the measurement of site-specific effects. A tissue residue approach was used in this Phase 1 ERA to evaluate risk from TBT to infaunal and epibenthic invertebrates. Risk to mobile, higher-trophic-level benthic invertebrates, as represented by Dungeness crab, was also characterized using the tissue residue approach. Estimated risks using the tissue residue approach are described in Section A.7.1.1.2.

A.7.1.1 Risk estimation

The risk estimation is divided into separate sections for sediment chemistry data (Section A.7.1.1.1), which are applicable to the risk characterization for benthic invertebrates that are closely associated with sediment, and tissue chemistry data (Section A.7.1.1.2), which are applicable to risk characterizations for TBT and for Dungeness crab.

A.7.1.1.1 Spatial analysis of predicted effects using sediment chemistry data

This section presents a spatial analysis of predicted effects, based on a comparison of sediment chemistry data to applicable standards and guidelines. All maps cited in this section are presented in Attachment A.1. The analysis focused on all COPCs rather than on any particular chemical group identified in the exposure assessment (Section A.3.1). The maps identify areas within the LDW where exceedances of sediment standards or guidelines have been observed. Exceedances of sediment standards or guidelines, particularly the CSL, represent a greater likelihood of adverse effects to benthic invertebrates than do samples without exceedances, but such exceedances do not necessarily provide definitive evidence that adverse effects have or

will occur at a particular location. The SMS rule provides the option of conducting biological testing to make such effects determinations.

The two COPCs with the greatest number of exceedances (see Table A-3-1) were total PCBs and BEHP; separate maps are provided for these two COPCs. The remainder of the analysis focuses on all 59 COPCs described in the exposure assessment (Section A.3.1). The maps discussed in this section portray Thiessen polygons so that areas of the site within various categories (e.g., < SQS/SL, > SQS/SL and <CSL/ML, > CSL/ML) can be calculated.

The maps in this section are based on detected values to calculate areas of exceedances. Specifically, if a chemical was not detected, detection limits were assigned a value of zero for display purposes (zero detection limit scenario), in contrast to the maps presented in the exposure assessment (Section A.3.1), which showed detected values and detection limits separately. Risk estimates based on detected values represented lower-bound estimates with the least uncertainty (i.e., they focus on areas known to exceed standards or guidelines based on detected concentrations). Incorporation of surrogate values for non-detects, such as one-half the detection limit, would introduce additional uncertainty for chemicals with detection limits above standards or guidelines. However, because concentrations could exceed sediment standards and guidelines in a greater area than identified assuming the zero detection limit scenario, maps using one-half the detection limit rather than zero for detection limits are also presented and discussed in the uncertainty assessment (Section A.7.1.2).

The percentages of the total study area associated with various concentration categories for PCBs and BEHP are shown in Table A-7-1. The data in this table come from Maps A-7-1 and A-7-2, which show concentrations of PCBs and BEHP, respectively, associated with Thiessen polygons. Additional discussion of the use of Thiessen polygons for spatial analysis is provided in Appendix C of the RI.

Table A-7-1. Percentage of total LDW area associated with concentration categories for PCBs and BEHP [a]

Chemical	Non-detect	<SQS	>SQS, <CSL	>1-5x CSL	5.1-10x CSL	10.1-20x CSL	>20x CSL
PCBs	6.5%	71%	18%	3.1%	0.96%	0.19%	0.050%
BEHP	28%	64%	4.4%	3.4%	0.040%	0%	0%

[a] Zero detection limit scenario

PCBs were detected in almost all samples; polygons associated with non-detections represent only 6.5% of the total area (Table A-7-1). Another 71% of the total area was associated with measured PCB concentrations below the SQS. Approximately 18% of the total area was above the SQS, but below the CSL, and less than 5% of the total area

was associated with CSL exceedances (Table A-7-1). CSL exceedance factors (EFs)[95] greater than 10 were rare (0.24% of total area, 13 locations). SQS exceedances were located throughout the LDW, but most CSL exceedances were located between Slips 4 and 6 in the eastern half of the LDW (Map A-7-1). Six polygons above the CSL were located upstream (south) of Turning Basin 3. Other locations with CSL exceedances were between RMs 1.9 and 2.3 (6 locations) and between RM 0.3 and 0.6 (8 locations).

BEHP was detected in most samples, but less often than PCBs; polygons associated with non-detections represent 28% of the total area (Table A-7-1). Another 64% of the total area is associated with measured concentrations below the SQS. Approximately 4.4% of the total area was above the SQS, but below the CSL, and less than 4% of the total area was associated with CSL exceedances (Table A-7-1). Most of the SQS and CSL exceedances were located between RMs 0.3 and 0.6 (Map A-7-2). Isolated CSL exceedances were located at RM 1.3-1.4, 1.9, 2.2, 3.5, 3.8, and 5.0 and in Slip 4.

The number of chemicals exceeding standards or guidelines at each sampling location is shown in Maps A-7-3 (SQS/SL) and A-7-4 (CSL/ML). Approximately 70%[96] of the total LDW area had no SQS/SL exceedances, and 91% of the total LDW area had no CSL/ML exceedances (Table A-7-2). Of the total LDW area, 7.2% had CSL/ML exceedances for a single chemical and 2.1% of the total LDW area had CSL/ML exceedances for two or more chemicals. SQS/SL exceedances were more uniformly spread throughout the LDW (Map A-7-3; approximately 30% of total area), but CSL/ML exceedances showed a more heterogeneous distribution (Map A-7-4). The greatest concentration of CSL/ML exceedances was found between RMs 0.3 and 0.6, and between Slip 4 and Slip 6. Smaller numbers of CSL/ML exceedances (6 or less) were seen in Slip 1, RM 1.3-1.4, RM 1.9, the vicinity of Slip 3, and upstream of Turning Basin 3 (Map A-7-4).

Table A-7-2. Percentage of total LDW area associated with number of chemicals exceeding SQS/SL and CSL/ML [a]

Standard or Guideline	0 COPCs	1-3 COPCs	4-7 COPCs	8-13 COPCs	>13 COPCs
SQS/SL	70%	27%	2.3%	0.54%	0.18%
	0 COPCs	**1 COPC**	**2-3 COPCs**	**>3 COPCs**	
CSL/ML	91%	7.2%	1.8%	0.31%	

[a] Zero detection limit scenario

The maximum SQS/SL and CSL/ML EFs (for any single chemical at each sampling station) are shown in Maps A-7-5 (SQS/SL) and A-7-6 (CSL/ML). Most maximum EFs

[95] Exceedance factor (EF) is the sediment concentration divided by the applicable standard or guideline. EFs have no regulatory relevance, and are not necessarily related to the degree of risk. They are presented here only to indicate the relative magnitude of chemical concentrations or detection limits.

[96] The areal percentages presented in Tables A-7-2 and A-7-3 are approximate because the number and shape of polygons differs between chemical based on sampling frequency. See RI Appendix C for additional details on Thiessen polygon methods.

were less than 5; EFs greater than 5 represented less than 7% and 2% of the total area with SQS/SL and CSL/ML exceedances, respectively (Table A-7-3). CSL/ML EFs greater than 10 represented less than 1% of the total area (19 locations). Most of the highest CSL/ML EFs were found either in Slip 4 or between RMs 3.1 and 3.6. Other areas with multiple CSL/ML EFs greater than 2 included between RMs 0.4 and 0.6, RM 2.2, and near Slip 6.

Table A-7-3. Percentage of total LDW area associated with maximum SQS/SL and CSL/ML exceedance factors (EF)[a,b]

Standard or Guideline	0 – 1 EF	>1 – 5 EF	>5 – 10 EF	>10 – 20 EF	>20 EF
SQS/SL	70%	23%	3.6%	1.3%	2.1%
	0 – 1 EF	**>1 – 2 EF**	**>2 – 5 EF**	**>5 – 10 EF**	**>10 EF**
CSL/ML	91%	4.8%	2.6%	1.4%	0.51%

[a] Zero detection limit scenario

[b] EFs have no regulatory relevance and are presented here to indicate the relative magnitude of measured concentrations.

The results of the spatial analysis suggest that there are large areas within the LDW where low risk to infaunal benthic invertebrates is predicted and other areas where risk is likely to be more significant. Approximately 70% of the LDW is predicted to pose low risk because of the lack of SQS/SL exceedances. An additional 10% of the LDW has a greater likelihood of adverse effects because of CSL/ML exceedances. The area between these two categories (approximately 20%) can be categorized as having an intermediate likelihood of adverse effects because of concentrations above the SQS/SL but below the CSL/ML.

Most of the locations with CSL/ML exceedances shown on Map A-7-4 were associated with exceedances of either total PCBs or BEHP. Map A-7-7 shows 37 locations with CSL/ML exceedances for other chemicals that did not also have CSL exceedances for either of these two chemicals. These locations are a subset of the locations shown on Map A-7-4. No single chemical was found at concentrations exceeding CSL/ML at more than 8 of the 37 locations shown on Map A-7-7. Several metals with CSL exceedances were found at several locations in the vicinity of RM 3.8. Organic chemicals with multiple CSL/ML exceedances not co-located with either total PCBs or BEHP included several PAHs, hexachlorobenzene, and 4-methylphenol. No spatial pattern for these exceedances is evident.

Because the AETs on which the standards and guidelines are based include several different endpoints, the standard or guideline exceedances for different chemicals do not predict a specific adverse effect on a site-wide scale. Potential adverse effects include mortality and abnormal development at the individual level and altered ecological function at the community level. Site-specific toxicity tests would reduce the uncertainty in predicting locations with adverse effects in benthic invertebrates. Among the data considered for the Phase 1 ERA, there were only a small number of toxicity tests from the LDW (Section A.3.2.2, Tables A-3-11 and A-3-12). A toxicity

testing approach will be proposed in the Phase 2 work plan to supplement the dataset of site-specific toxicity test results, and risks to benthic invertebrates will be further evaluated in the Phase 2 ERA consistent with the SMS framework. These toxicity test results should reduce uncertainties in the benthic risk conclusions to allow for more informed risk management decisions.

A.7.1.1.2 Analysis based on tissue chemistry

In the previous section, risks to benthic invertebrates were primarily addressed using site-specific sediment chemistry data and estimated sediment-based effect concentrations (i.e., SMS standards and DMMP guidelines). In this section, a tissue-based approach was used to assess risk to crab from all COPCs (Crab Assessment subsection, below) and to benthic invertebrates from TBT (TBT and Benthic Invertebrates subsection, below). A tissue-based approach was more appropriate for crab because they are more mobile than infaunal organisms, are not specifically covered by SMS metrics, and have a greater potential for exposure through bioaccumulation because of their higher trophic position. For TBT, a tissue-based approach is generally preferable for ERA purposes (EPA 1999; Meador 2000), although measurements in porewater are the basis for DMMP guidelines for TBT.

Crab Assessment

A summary of exposure and effects data to evaluate risks to crab was previously presented in Tables A-3-17 and A-3-18 in Section A.3.3. In this section, HQs are calculated using these data (Table A-7-4), to assess whether COPCs may be posing risks to crab based on existing data.

The only HQ greater than 1 for crab was a NOEC-based HQ of 10 for arsenic, based on a crayfish study (Table A-7-4). For chromium, mercury, and zinc, the NOEC- and LOEC-based HQs were less than 1. Copper, PCBs, and TBT NOEC-based HQs were also all less than 1 (LOEC TRVs were not available for these chemicals). Uncertainties in this assessment are discussed in Section A.7.1.2.2, and risk conclusions are discussed in Section A.7.1.3.2.

Table A-7-4. Crab HQs using hepatopancreas and whole-body exposure and effects data

Chemical	HQ–Hepatopancreas		HQ–Whole Body[a]	
	NOEC	LOEC	NOEC	LOEC
Arsenic	na	na	**10**	na
Cadmium	na	na	0.008[b]	0.0004[b]
Chromium	na	na	0.15	0.05
Copper	na	na	0.59[c]	na
Mercury	0.68	0.67	na	na
Zinc	0.45	0.22	na	na
PCBs	na	na	0.02	na
TBT	na	na	0.65	na

Note: HQs greater than 1 are noted in **bold type**.

na – Not available

[a] Whole body concentrations were estimated by combining hepatopancreas and edible meat concentrations, assuming 85% by mass edible meat and 15% by mass hepatopancreas.

[b] Based on effect concentration in muscle tissue

[c] Based on effect concentration in claw tissue

TBT and Benthic Invertebrates

In this section, potential adverse effects to benthic invertebrates from exposures to sediment-associated TBT are assessed. Using a tissue residue TRV of 3 mg/kg dw and estimated (as described in Section A.3.1.2.2) and measured TBT concentrations in tissue, HQs were calculated for the range of tissue data (Table A-7-5).

Table A-7-5. HQs calculated for TBT using measured and estimated tissue concentrations

Basis	HQ[a]
Measured maximum tissue concentrations near Kellogg Island	0.06
Tissue concentration estimated using:	
Minimum sediment concentration	0.008
Median sediment concentration	0.30
Maximum sediment concentration	**5.3**

Note: HQs greater than 1 are noted in **bold type**.

[a] Based on comparison of the tissue concentration to a threshold (3 mg/kg dw) for effects on survival, growth, and reproduction data for benthic invertebrates (EPA 1999b; Meador 2000). See Section A.3.2.

HQs less than 1 (0.06 to 0.30) were calculated for TBT and benthic organisms using measured tissue concentrations and tissue concentrations estimated using the median concentration in sediment in the LDW. However, when the maximum TBT sediment concentration from the LDW was used to estimate a tissue concentration for comparison to the effects benchmark, the HQ was 5.3, thus indicating the potential for

adverse effects in the LDW. The highest concentrations of TBT were located in the lower 2 miles of the LDW (Map A-7-8).

A.7.1.2 Uncertainty assessment

In this section, uncertainties identified in the risk estimates for benthic invertebrates are discussed, including the use of SMS to estimate risk to benthic invertebrates. Issues associated with the availability and interpretation of effects and exposure data used in the tissue residue approach used for crab and for TBT and benthic invertebrates are also discussed.

A.7.1.2.1 Benthic invertebrates and sediment chemistry

This section presents uncertainties relevant to the assessment of infaunal and epibenthic benthic invertebrates using surface sediment chemistry. Uncertainties are discussed separately for the problem formulation, exposure assessment, and effects assessment.

Problem Formulation

Lack of standards and guidelines for all chemicals

AETs, which form the basis for SMS standards and DMMP guidelines, exist for only about 20% of the chemicals that have been measured in the LDW. Chemicals without standards or guidelines were not identified as COPCs in the problem formulation. Because the risk characterization for benthic invertebrates is based primarily on a spatial analysis of chemicals from many different chemical groups, it is likely that the locations with the highest potential for adverse effects were adequately identified using the existing standards and guidelines. Consequently, the lack of appropriate standards or guidelines for many chemicals is likely to have a low impact on the overall risk conclusions, although the uncertainty associated with risks from chemicals without standards or guidelines is high.

Exposure Assessment

Use of surface sediment chemistry data to characterize animals that burrow > 15 cm

Some benthic invertebrate species (e.g., clams) may burrow deeper than the 15-cm surface sediment threshold used in the Phase 1 ERA. These animals may be exposed to subsurface sediments that have not been characterized in this ERA. A risk characterization that included these animals could have a different outcome than the risk characterization presented in Section A.7.1.1.1 if either of two conditions were met: 1) concentrations in sediment > 15 cm were markedly different from concentrations in sediment < 15 cm; or 2) the chemical sensitivity of animals living below 15 cm is markedly different than the chemical sensitivity of animals on which the existing chemical standards and guidelines are based. Because few site-specific data exist regarding the benthic community and chemical concentrations at depths greater than 15 cm, the utility and feasibility of collecting clams for tissue analysis, benthic community analyses to assess community structure, and additional sediment

core samples will be discussed in the data gaps memorandum. These data would provide additional information related to exposure in sediments deeper than 15 cm.

Sampling density for certain COPCs

Most COPCs have been measured in surface sediment at >500 locations, but some, such as total DDTs (102 locations) and TBT (94 locations), have been analyzed at fewer locations (see Section A.3.1). The certainty of risk characterization for these chemicals is lower compared to chemicals measured more frequently. However, because all COPCs were analyzed at least 50 locations, the potential impact of this uncertainty on overall risk conclusions is likely to be low. The necessity and feasibility of collecting additional data for some chemicals that have been analyzed less frequently will be evaluated in Phase 2.

Treatment of detection limits for spatial analysis

Sediment chemistry data typically include many records where a reliable concentration could not be determined, i.e., a non-detection. The maps and areal percentages of SMS exceedance presented in Section A.7.1.1.1 were based on the assumption that concentrations of chemicals not detected above analytical detection limits were zero. An alternate data analysis method for detection limits is presented in this section. It is common in risk assessments to assume a value of one-half the detection limit to represent the concentration of undetected chemicals. Therefore, for illustration purposes, the maps and areal percentages of SMS exceedance presented in Section A.7.1.1.1 were also calculated using a value of one-half the detection limit for undetected chemicals rather than treating the concentration of undetected chemicals as zero. The recalculated percentages are discussed below.

In Maps A-7-9 and A-7-10, one-half detection limits for PCBs and BEHP were compared to SQS and CSL and mapped accordingly. In contrast, no such comparison was made in Maps A-7-1 or A-7-2 because non-detections were assumed to be zero. For PCBs and BEHP, one-half detection limit-based values exceeded the SQS for only a very small number of samples, as reflected by the slight increase in areal percentages in the ">SQS, <CSL" category (Table A-7-6). The areal percentage of CSL exceedances was identical regardless of how detection limits were treated, indicating that all the analytical detection limits were below the CSLs.

Table A-7-6. Percentage of total LDW area associated with concentration categories for PCBs and BEHP (zero and half detection limit scenarios)

Chemical	Non-detect	<SQS	>SQS, <CSL	1-5x CSL	5.1-10x CSL	10.1-20x CSL	>20x CSL
PCBs (zero for non-detects)	6.5%	71%	18%	3.1%	0.96%	0.19%	0.050%
PCBs (half detection limit)	na	78%	18%	3.1%	0.96%	0.19%	0.050%
BEHP (zero for non-detects)	28%	64%	4.4%	3.4%	0.040%	0	0
BEHP (half detection limit)	na	92%	4.4%	3.6%	0.040%	0	0

na – Not applicable

The differences in areal percentages between the two methods were greater with respect to the number of chemicals exceeding standards or guidelines compared to the chemical-specific comparisons presented in Table A-7-6. The one-half detection limit scenario increased the total area with one or more SQS/SL exceedances from 30% to 63% (Table A-7-7). The total area with CSL/ML exceedances increased from 9.3% to 14%. The additional areas with exceedances were attributed to elevated detection limits for many semi-volatile organic compounds. The exceedance frequency for many of these compounds was much higher for detection limits compared to detected concentrations (see Table A-3-1). The elevated detection limits were not centered on a particular area (Maps A-7-11 and A-7-12). A similar increase in areal percentages as a function of detection limit treatment was noted for maximum EFs (Table A-7-8). As with the numbers of chemicals, the additional areas with exceedances were spread throughout the LDW (Maps A-7-13, A-7-14).

Table A-7-7. Percentage of total LDW area associated with number of chemicals exceeding SQS/SL and CSL/ML (zero and half detection limit scenarios)

Standard or Guideline	0 COPCs	1-3 COPCs	4-7 COPCs	8-13 COPCs	>13 COPCs
SQS/SL (zero for non-detects)	70%	27%	2.3%	0.54%	0.18%
SQS/SL (half detection limit)	37%	53%	6.1%	1.7%	2.3%
	0 COPCs	**1 COPC**	**2-3 COPCs**	**>3 COPCs**	
CSL/ML (zero for non-detects)	91%	7.2%	1.8%	0.31%	
CSL/ML (half detection limit)	86%	8.00%	2.4%	3.1%	

Table A-7-8. Percentage of total LDW area associated with maximum SQS/SL and CSL/ML EFs[a] (zero and half detection limit scenarios)

STANDARD OR GUIDELINE	0 – 1 EF	>1 – 5 EF	>5 – 10 EF	>10 – 20 EF	>20 EF
SQS/SL (zero for non-detects)	70%	23%	3.6%	1.3%	2.1%
SQS/SL (half detection limit)	37%	53%	4.8%	1.5%	4.1%
	0 – 1 EF	**>1 – 2 EF**	**>2 – 5 EF**	**>5 – 10 EF**	**>10 EF**
CSL/ML (zero for non-detects)	91%	4.8%	2.6%	1.4%	0.51%
CSL/ML (half detection limit)	86%	6.9%	2.3%	1.9%	2.5%

[a] EFs have no regulatory relevance and are presented here to indicate the relative magnitude of measured concentrations.

The comparison between the methods of treating non-detected chemicals suggests that using one-half the detection limit identified more areas as potentially exceeding standards or guidelines. However, any such additional areas would have high uncertainty associated with the actual concentration of undetected chemicals. Many of the samples for which a value of one-half the detection limit exceeded the SQS or CSL likely had artificially high detection limits that may have been attributable to interferences in the analytical method. It is generally possible to achieve detection limits lower than the SQS. Therefore, the overall uncertainty associated with the use of zero for non-detects is medium, and the potential impact on risk conclusions is low because chemicals with more frequent exceedances based on detected concentrations are likely to drive any risk-based decisions. The utility and feasibility of collecting additional data for chemicals with detection limits above applicable standards or guidelines will be evaluated in Phase 2.

Effects Assessment

Limited site-specific biological effects data

For the Phase 1 ERA, very few LDW-specific data exist regarding direct assessment of biological effects on benthic invertebrates (see Section A.3.2, Tables A-3-11 and A-3-12). Because there are so few data, they cannot be used to characterize system-wide effects for this group of receptors. As discussed in Section A.3.2.2, the existing site-specific toxicity test data evaluated for the Phase 1 ERA indicated low risk to benthic invertebrates. Nine of the ten samples passed the biological effects criteria of the sediment quality standards, even though seven of those samples had at least one chemical CSL exceedance and one other sample had two chemical SQS exceedances. However, most areas of the LDW have not been characterized in this manner. Consequently, the system-wide risk estimates in the Phase 1 ERA are based on predictions of biological effects on benthic invertebrates using chemistry alone. Additional biological effects data could be used to provide a direct assessment of biological effects for localized areas with SQS or CSL exceedances. Additional sediment toxicity tests will be conducted in Phase 2 to reduce this uncertainty.

Uncertainty in using SMS for effects assessment and risk characterization

The likelihood of adverse effects on benthic organisms was assessed based on a comparison of site-specific surface sediment chemistry data to SMS standards or DMMP guidelines, as specified in the SOW.[97] The toxicity test species included in these standards and guidelines represent only a small portion of the diverse benthic invertebrate community present in the LDW. However, the benthic community AET, which is used to set several SQS or CSL values, does incorporate many different species and biological mechanisms. Nonetheless, potential effects to some LDW benthic species may not be addressed by these standards and guidelines; therefore, a risk characterization based on them may not represent potential effects to the entire benthic community. Consequently, there is some uncertainty associated with the risk estimates made from these standards and guidelines. As with any surrogate, certain assumptions regarding relative sensitivities among species were required.

Sediment quality guidelines (SQGs) have been developed by several researchers (e.g., Long et al. 1995; MacDonald et al. 1996; Smith et al. 1996). Although the SMS standards (SQS and CSL) may not accurately reflect potential adverse effects to all benthic invertebrate species in the LDW, this uncertainty is not unique to the SMS. Comparisons to SQGs other than the SMS may yield different predictions from those presented in Section A.7.1.1.1, but risk predictions made from other SQGs may be equally uncertain. While such quantitative comparisons are not presented in this uncertainty assessment, background information on the various SQGs is presented here to qualitatively assess the level of uncertainty inherent in these alternatives.

Various theoretical and empirical approaches have been used to derive SQGs. MacDonald et al. (2000) described the 5 most widely applied approaches: 1) equilibrium partitioning, 2) screening-level concentration, 3) effects range, 4) effects level, and 5) AETs. These approaches are compared in Table A-7-9.

An important issue to consider when evaluating the uncertainty of the various SQGs is the biological endpoints used to develop the guidelines. The benthic invertebrate species assemblage in the LDW more closely reflects benthic communities in Puget Sound (see Section A.2.2.2). Accordingly, marine and estuarine SQGs are probably most applicable for this site. The database constructed by Long et al. (1995) contains more endpoints than the database used to derive the Puget Sound AETs (PTI 1988). However, some of the data used by Long et al. (1995) are not necessarily applicable to the assessment of benthic invertebrate health because the data set also includes non-benthic invertebrate endpoints (e.g., histopathological disorders in demersal fish). As originally configured, the ER-L and ER-M developed by Long et al. (1995) were designed to assess sediment quality in general, not necessarily benthic invertebrate health in the context of an ERA.

[97] The SOW also specified the use of tissue data, where appropriate.

Table A-7-9. Comparison of approaches for developing SQGs

Approach	SQG Abbreviation	Basis	Tests/Endpoints	Sediment Type	Reference
Screening level concentration	SLC	empirical	Presence of specific benthic invertebrate species	freshwater	NY DEC 1999
				freshwater	Persaud et al. 1993
Effects range	ER-L, ER-M	empirical and theoretical	Benthic communities, individual invertebrate species used for sediment toxicity tests, histopathological observations of fish, spiked sediment toxicity tests, equilibrium partitioning models	marine/ estuarine	Long et al. 1995
			Hyalella azteca and *Chironomus tentans* sediment toxicity tests	freshwater	EPA 1996
Effects level	TEL, PEL	empirical and theoretical	Sediment toxicity tests for many invertebrate species	marine	MacDonald et al. 1996
			Hyalella azteca and *Chironomus tentans* sediment toxicity tests	freshwater	Ingersoll et al. 1996
			Sediment toxicity tests for many invertebrate species	freshwater	Smith et al. 1996
Apparent Effects Threshold	AET	empirical	Microtox®, oyster larvae, and amphipod sediment toxicity tests, benthic community analyses	marine/ estuarine	PTI 1988
			Many invertebrate species and Microtox® sediment toxicity tests	freshwater	Cubbage et al. 1997
Equilibrium partitioning	SQG	theoretical	Many invertebrate and fish species upon which ambient water quality criteria are based	marine and freshwater	EPA 1993a

Recently, researchers have combined multiple SQGs to develop "consensus" SQGs for PCBs, metals, PAHs, and pesticides (MacDonald et al. 2000) and PAHs (Swartz 1999), and mean SQGs for multiple chemicals as indicators of amphipod toxicity (Fairey et al. 2001). These aggregated SQGs were derived in part by evaluating their ability to predict adverse effects. Hyland et al. (1999) demonstrated an increased probability of adverse benthic community impacts as mean HQs increase (relative to SQG values).

SQGs and their relationship to sediment quality and risk assessment are being widely debated within the scientific community. There is growing concern within the scientific community that undue emphasis has been given to the application of numerical guidelines to evaluate sediment contamination and to formulate risk management decisions. Accordingly, a Pellston workshop[98] on sediment quality assessment took place in August 2002. The following aspects of the current debate on SQGs were explored at that workshop:

[98] Pellston workshops are sponsored by the Society of Environmental Toxicology and Chemistry (SETAC) and are intended to convene recognized experts in particular disciplines to discuss and debate scientific issues pertinent to the Society's interests. The proceedings of such workshops are typically published.

- What are the scientific underpinnings and uncertainties associated with SQGs?
- How well do SQGs represent the potential for effects or no effects on aquatic biota?
- How can SQGs be used in one or more frameworks to assess sediment contamination?
- How should other assessment tools available for evaluating sediment contamination be used in combination with SQGs?
- What are the assessment and management options for addressing particularly complex sediment systems?

When available, the results of this workshop may provide useful information for future phases of this project.

Predictiveness

A fundamental distinction must be made in risk assessments between measured and predicted effects. Because of limited available data on site-specific biological effects, the risk characterization for sediment-associated benthic invertebrates was based on predicted effects using available sediment chemistry standards and guidelines. The degree to which the predicted effects are meaningful depends on the predictiveness of these standards and guidelines, which in turn depends on site-specific conditions that affect chemical bioavailability, as well as other conditions such as non-chemical stressors.

SMS standards and DMMP guidelines were developed using the AET approach described in Section A.3.3. Given the small number of toxicity tests and benthic macroinvertebrate samples, site-specific AETs were not developed because comparison of sediment chemistry data from the samples tested in laboratory toxicity tests with the applicable AETs presented in Section A.3.2.1 would be inconclusive. However, there has been an evaluation of the predictiveness of select AETs in the scientific literature; this analysis is summarized below.

Gries and Waldow (1996) examined the predictiveness of some AETs using a Puget Sound-wide data set. They assessed reliability of the 1994 amphipod and echinoderm AETs using three measures: sensitivity, efficiency, and overall reliability. Sensitivity was defined as the percentage of correctly predicted "hit"[99] samples. Efficiency was calculated as the percentage of all predicted "hit" samples that exhibited significant adverse effects. Overall reliability was calculated as the percentage of all "hit" and "no hit" samples that were correctly predicted. Table A-7-10 shows the sensitivity and overall reliability measurements for amphipod and echinoderm AETs.

[99] Those samples that exceeded at least one AET value and also exhibited significant adverse biological effects.

Table A-7-10. The predictive reliability of amphipod mortality and echinoderm larvae abnormality AETs

AET Group	Database for Comparison	Total Samples	Sensitivity	Efficiency[a]	Overall Reliability
Dry-weight normalized					
1994 Amphipod AETs	1994 Amphipod	674	43% (79/181)	100% (79/79)	84% (579/674)
1988 Amphipod AETs	1994 Amphipod	674	48% (87/181)	38% (87/227)	65% (438/674)
1988 Amphipod AETs	1988 Amphipod	287	56% (59/106)	100% (62/62)	85% (243/287)
1994 Echinoderm AETs	1994 Echinoderm	205	48% (38/79)	100% (38/38)	80% (164/205)
TOC normalized					
1994 Amphipod AETs	1994 Amphipod	478	36% (58/162)	100% (58/58)	78% (368/478)
1988 Amphipod AETs	1994 Amphipod	671	34% (61/181)	46% (61/133)	71% (476/671)
1988 Amphipod AETs	1988 Amphipod	287	45% (48/106)	100% (48/48)	80% (229/287)
1994 Echinoderm AETs	1994 Echinoderm	205	46% (36/79)	100% (36/36)	79% (162/205)

Source: Table 7 in Gries and Waldow (1996)

[a] When comparing AETs against the database from which they were derived, efficiency is always equal to 100% because of the way in which AETs are calculated (Gries and Waldow 1996).

The analyses conducted by Gries and Waldow (1996) indicated that the sensitivity of the amphipod and echinoderm AETs was generally 50% or less with overall reliabilities of 70-80%. TOC normalization appeared to make little difference in the overall AET reliability. Calculations similar to those presented in Table A-7-10 could not be made for the available LDW data (post 1990) because there were only ten amphipod test results (with no "hits") and only seven echinoderm test results (with only a single "hit"). The LDW data are described in Sections A.3.2.2 and A.3.2.3.

There is medium uncertainty in risk estimates made from AET-based standards and guidelines because of the limited site-specific biological effects data available to validate these estimates. Additional effects data would likely have a medium impact on risk conclusions, but only a low probability of increasing risk estimates. It is more likely that additional biological effects data would reduce areas that are now identified as SMS exceedances.

A.7.1.2.2 Benthic invertebrates and tissue chemistry

This section presents uncertainties relevant to the crab assessment and the TBT assessment for benthic invertebrates. Both of these assessments used a tissue-based approach.

Crab Assessment

This section presents specific areas of uncertainty in the crab assessment related to the problem formulation, exposure assessment, and effects assessment.

Problem formulation

Crabs were selected as an ROC to represent higher trophic-level benthic invertebrates not covered by the SMS. There is some uncertainty associated with the assumption

that COPC concentrations in crab tissue would represent those of other mobile, higher-trophic-level benthic invertebrates in the LDW, which include sea stars and shrimp. Dungeness crabs are scavengers, with a diet including shrimp, mussels, small crabs, clams, and sea urchins. Thus, crabs are likely to be similarly exposed through their diet as sea stars and shrimp, which have a similar diet.

Exposure assessment

Because tissue was used to estimate exposure to crab, all potential exposure pathways were integrated. However, because the crab home range can include areas outside of the LDW, the tissue burden may not be fully reflective of LDW exposure.

A small number of crab tissue data were available (Section A.3.1.2.1), and samples were collected from the lower waterway in the vicinity of Kellogg Island. Although crabs could potentially encounter higher concentrations of sediment-associated COPCs, such as PCBs, in other parts of the river, the extent to which crabs use the area at locations upstream of RM 2.0, where salinity is lower, is unknown. Although collection of crab samples in the LDW as far upstream as RM 4 was attempted in 1998, no adult crabs were observed or caught (ESG 1999), although juveniles were collected up to the 1st Avenue bridge (RM 2.1). Several crab species were also caught in PSAMP otter trawls, including graceful crab, Dungeness crab, porcelain crab, and Oregon cancer crab in the LDW between RMs 0 and 1.5. Estimated whole-body concentrations in crab would need to increase by nearly two orders of magnitude (from 0.40 to 23 mg/kg ww) for the NOEC-based PCB HQ, for example, to exceed 1. This seems unlikely, regardless of where the crabs are caught within the LDW.

Toxicological data were available for six chemicals that were not analyzed in crab tissue. These chemicals were chlordecone, DDT, methoxychlor, mirex, 1,2,3-trichlorobenzene, and 1,2,3,4-tetrachlorobenzene. Crabs were not analyzed for any pesticides, so potential effects associated with exposure to these chemicals are not known.

There is uncertainty associated with the LDW whole-body crab tissue residue data because they were estimated based on chemical concentrations in edible meat (total of 6 crab composite samples available) and the hepatopancreas (1 composite sample available). It is unknown if whole-body concentrations estimated from these data result in an over- or under-estimate of the actual whole-body exposure of crabs to COPCs due to the limited dataset available. In total, the uncertainty associated with the limited crab tissue dataset is high.

Effects assessment

The primary uncertainty in the crab effects assessment is the limited amount of relevant tissue-effect data available in the literature. Effects data were found only for eight of the COPCs detected in LDW crab tissue. These toxicity studies investigated only survival or growth endpoints, although it is possible that reproductive endpoints might be more sensitive. For half of the COPCs (cadmium, copper, mercury, and zinc),

tests were done with adults only, although juvenile or early life stages may be more vulnerable than adults to body burdens accumulated through dietary exposure. Additional uncertainty associated with these study methods include exposure duration, exposure pathway (water exposure vs. dietary), test organism used (other crab species or other decapods), and measurement of residues in tissue (such as claw tissue) other than hepatopancreas or whole body. Uncertainties associated with toxicity studies for each COPC are summarized in Table A-7-11. In particular, effects data for copper were highly uncertain because of the short exposure time of the toxicity study, the water exposure pathway, and analysis of copper only in crayfish claw tissue. Effects data for decapods were not found for a number of chemicals measured in crab tissue, including lead, nickel, silver, and numerous semivolatile or volatile compounds[100], so risk from body burdens of these COPCs cannot be addressed.

For some COPCs, only NOEC TRVs were available. For arsenic, which had a NOEC-based HQ exceeding 1 but no LOEC-based TRV, it was not possible to determine if effects would be observed at measured arsenic concentrations (i.e., the threshold corresponding safe concentration was not known because no effects have been observed in any toxicological studies with arsenic and decapods). Therefore, the NOEC-based HQ exceeding 1 does not necessarily indicate a risk. In total, crab or decapod toxicity data have a high level of uncertainty, and could lead to under- or overestimation of risk to crab, with a medium impact on risk conclusions.

Table A-7-11. Factors contributing to uncertainty for TRV studies selected as NOECs and LOECs for crabs

TRV	Short Length of Exposure	Water-only Exposure Pathway	Data for Decapods other than Crab	Adult Life Stage	NOEC Only	Muscle or Claw Tissue Only
Arsenic	X	X	X		X	
Cadmium		X		X		X[a]
Chromium		X				
Copper	X	X	X	X	X	X[b]
Mercury			X	X		
Zinc	X	X	X	X		
PCBs	X	X			X	
TBT	X				X	

Factors contributing to uncertainty are identified by X

[a] muscle tissue

[b] claw tissue

[100] All of the semivolatile and volatile compounds, except PCBs and benzyl alcohol, were undetected in crab tissue.

TBT Assessment

The assessment of potential impacts from TBT was based on measured or estimated tissue residues in benthic invertebrates rather than sediment chemistry. Key uncertainties in this assessment are primarily related to the exposure and effects assessments, as described below.

Exposure assessment

Four composite benthic invertebrate tissue samples from the LDW have been analyzed for TBT (King County 1999c). These samples were collected near Kellogg Island, and constituted approximately 87% *Eogammarus* (an epibenthic amphipod) and 13% *Corophium* (an infaunal amphipod). Using this relatively small dataset to assess potential TBT exposure of benthic invertebrates is uncertain because:

- it was a small dataset (n=4) of composite samples of approximately 2,000 organisms each
- only two types of benthic invertebrate feeding groups were represented, and the majority are epibenthic amphipods
- the highest sediment TBT concentrations were not located in the vicinity of amphipod collection sites near the west side of Kellogg Island
- site usage of neo- and mesogastropods (the taxa most susceptible to effects of TBT) in the LDW is uncertain, and, if present, it is unknown what their TBT tissue residues might be

To address uncertainty associated with collection of tissue samples near Kellogg Island, TBT concentrations were estimated at other areas in the LDW using a modified BSAF calculated from the limited available data (i.e., modified BSAFs were applied to median and maximum TBT concentrations in sediment to estimate median and maximum tissue concentrations). As shown in Map A-7-8, the highest TET concentrations were located in the lower 2 miles of the LDW, primarily in subtidal habitat, except for a single station near RM 3.3.

While use of a modified BSAF provides some indication of potential tissue concentrations in areas with higher sediment TBT concentrations, it is itself highly uncertain because of the:

- limited dataset from which the modified BSAF was derived, both in terms of types of species and sample numbers
- potentially different TBT bioavailability in other areas of the LDW

The high uncertainty associated with the estimated TBT concentrations has a high impact on the risk conclusions. Although HQs associated with measured concentrations of TBT in tissues and HQs associated with estimated median sediment concentrations of TBT in tissues are less than 1, HQs associated with 18 of the 102 stations analyzed for TBT could be greater than 1 based on tissue concentrations

estimated using a modified BSAF. The feasibility and utility of addressing this data gap will be discussed in the data gaps memorandum.

Effects Assessment

The tissue residue concentration selected for assessment of risk associated with TBT in invertebrate tissues was primarily based on the growth endpoint (Meador 2000), although this threshold is supported by toxicological data related to the survival endpoint and certain reproductive endpoints as well (EPA 1999). TBT exposure is also associated with two sublethal effects specific to a small group of species, bivalve shell thickening in oysters and induction of imposex or intersex[101] in gastropod snails. Shell thickening in oysters was not recommended as an endpoint, however, due to the lack of oyster populations and suitable habitat for oysters in the LDW. Potential impacts to gastropod snails are discussed below.

Members of the orders Mesogastropoda and Neogastropoda have been shown to be sensitive to imposex resulting from TBT exposure. Tissue residue concentrations associated with sterilization due to imposex are available for three species (Table A-3-17; *Littorina littorea, Ocinebrina aciculata, Nucella lapillus*). It should be noted that tissue residue concentrations associated with the onset of imposex are considerably lower than those associated with complete sterilization.

Periwinkle (*Littorina littorea*) is found along coasts and estuaries in Europe from northern Spain to the White Sea (northern Russia) as well as the northeastern US and eastern Canada. Littorina species found in the Pacific Northwest include checkered periwinkle (*Littorina scutulata*) and grey periwinkle (*Littorina planaxis*). Both of these species prefer rocky intertidal habitat. No *Littorina* species have been reported to be present in the LDW. Dogwhelk (*Nucella lapillus*) is generally found along the coasts and estuaries in Europe, Greenland and the Northeastern US and eastern Canada. *Nucella* are found in Puget Sound (Dethier and Schoch 2000), but do not likely occur in the LDW due to lack of appropriate hard substrate. However, four species of the order Neogastropoda and five species of the order Mesogastropoda have been collected in the LDW (see Table A-2-2). Further investigation would be required to determine site usage of gastropod species particularly sensitive to TBT (or potentially present based on habitat) in the LDW.

A.7.1.2.3 Summary of uncertainties for benthic invertebrates

Table A-7-12 summarizes the uncertainties related to exposure of benthic invertebrates and crab, and ranks the uncertainties according to level of uncertainty (as low, medium, or high), potential to impact the risk conclusions, and feasibility to fill data gaps. This context is helpful in understanding the risk estimate and the probability

---

[101] As stated in EPA (1999), imposex is defined as the development of male sexual characteristics in females. Intersex is characterized as any disturbance of phenotypic sex determination between gonad and genital tract (see Bauer et al. 1997).

that the estimate is predictive of risk. For example, a high potential impact on risk conclusions suggests a high probability that estimates are either over- or underestimating risk and that the difference may be enough to change the conclusions.

Concentrations of TBT in neo- and mesogastropods are highly uncertain and could have a high impact on the risk conclusion. In the crab assessment, tissue residue data and site usage information for crabs are viewed as having a medium impact on risk conclusions, primarily because the effects data for crab were also uncertain. The uncertainty associated with use of sediment standards and guidelines to predict risk on a site-specific basis was also categorized as medium, due to the low number of sediment toxicity tests conducted in the LDW. Additional sediment toxicity tests will be needed to assess site-specific toxicity using an approach to be outlined in the Phase 2 work plan.

Table A-7-12. Summary of uncertainties associated with benthic invertebrate risk characterization

Issue	Level of Uncertainty	Effect of Uncertainty on Risk Estimate	Potential Means to Decrease Uncertainty	Potential Impact on Risk Conclusions	Feasibility of Filling Data Gap
Exposure Assessment					
Coverage of sediment data in the LDW for selected COPCs (e.g., DDT and TBT)	medium	unknown[a]	collect additional surface sediment samples	low	high
Use of surface sediment chemistry data to characterize exposure to benthic invertebrates	low	unknown[a]	collect relevant exposure samples	low	high
Use of zero detection limit for risk characterization	medium	possible under-estimation	collect additional sediment with lower DLs	low	medium
TBT tissue concentrations in benthic invertebrates	high	unknown[a]	collect tissue data in areas across a TBT gradient	high	high
Suitable habitat for crabs	medium	possible under-estimation	conduct a site usage survey	medium	high
Use of crabs to represent other ROCs in LDW from an exposure perspective	medium	unknown[a]	collect other organism from similar trophic position[b]	medium	low
Use of limited crab tissue dataset	high	unknown[a], but possibly under-estimated	collect additional crab tissue	medium	high

Issue	Level of Uncertainty	Effect of Uncertainty on Risk Estimate	Potential Means to Decrease Uncertainty	Potential Impact on Risk Conclusions	Feasibility of Filling Data Gap
Effects Assessment					
Use of SMS standards and DMMP guidelines to estimate site-specific effects to benthic invertebrates	low	unknown[a]	conduct additional toxicity tests with LDW sediment	medium	medium
Crab toxicity data	high	unknown[a]	conduct additional toxicity tests with crabs	medium	low
Use of crab as a representative of other higher-trophic-level benthic species	medium	unknown[a]	conduct toxicity test with other species; or conduct a literature search on relative sensitivities	low	low; medium

[a] Effect is dependent on whether additional exposure or TRV data would have higher or lower COPC concentrations than existing data.

[b] Relevant toxicological data would need to be available to assess risks.

Level of uncertainty key: **low** = large and relevant dataset
medium = small dataset or limited information
high = very limited data or no site-specific information

Potential impact key: **low** = unlikely to result in a change of HQ from less than 1 to greater than 1 (or vice versa)
medium = could result in a change of HQ from less than 1 to greater than 1 if worst-case scenario is used (scenario is viewed as unlikely)
high = HQ could change from less than 1 to greater than 1 (or vice versa) using a scenario that is conservative but more reasonable than the worst-case scenario

Feasibility key: **low** = high budget or difficult research study would be required to address uncertainty
medium = issue could be resolved with a mid-level field sampling event or research study or a detailed assessment of literature
high = issue could be resolved with additional literature search or through limited field sampling

Table A-7-13 summarizes the primary uncertainties related to the crab TRVs and ranks them according to level of uncertainty, and the potential to impact the risk conclusions. Although uncertainties are ranked medium to high for all COPCs, the impact on risk conclusions is low for some COPCs, such as cadmium and PCBs, due to very low HQs (Table A-7-4).

Table A-7-13. Summary of uncertainties associated with TRVs used in crab risk characterization

TRV	Level of Uncertainty	Potential Impact on Risk Conclusions
Arsenic	high	high
Cadmium	high	low
Chromium	medium	medium
Copper	high	high
Mercury	medium	medium
Zinc	high	medium
PCBs	medium	low
TBT	medium	medium

Level of uncertainty key: **low** = large or relevant dataset
medium = small dataset or limited information
high = very limited data

Potential impact key: **low** = unlikely to result in a change of HQ from less than 1 to greater than 1 (or vice versa)
medium = could result in a change of HQ from less than 1 to greater than 1 if worst-case scenario is used (scenario is viewed as unlikely)
high = HQ could change from less than 1 to greater than 1 (or vice versa) using a scenario that is conservative but more reasonable than the worst-case scenario

A.7.1.3 Risk conclusions

This section synthesizes the risk estimation and uncertainty results for benthic invertebrates. Risk to benthic invertebrates was characterized using existing data and based on a spatial analysis of all COPCs[102] relative to sediment standards and guidelines. No benthic invertebrate COPCs were eliminated in Phase 1. TBT was evaluated based on tissue chemistry. Risks from all COPCs to crab were also evaluated based on tissue chemistry. Risk conclusions for benthic invertebrates are described in more detail below.

A.7.1.3.1 Benthic invertebrates and sediment chemistry

Locations where potential adverse effects to sediment-associated invertebrates were predicted by comparing existing surface sediment chemistry data to SMS numeric standards and DMMP guidelines. Approximately 70% of the LDW was predicted to pose low risk to benthic invertebrates because of the lack of SQS/SL exceedances. An additional 10% of the LDW was predicted to have a greater likelihood of adverse effects because of CSL/ML exceedances. The area between these two categories (approximately 20% of the LDW) can be characterized as having an intermediate likelihood of adverse effects because of concentrations above the SQS/SL but below the CSL/ML. The potential adverse effects included in the existing standards are mortality and abnormal development at the individual level and altered ecological function at the community level. Additional toxicity tests would be required to reduce uncertainty in risk predictions.

Exposure to the 59 chemicals identified in the problem formulation as benthic COPCs using existing data and sediment standards or guidelines[103] was assessed in the exposure assessment by grouping the COPCs into categories based primarily on the frequency and magnitude of sediment standard or guideline exceedance (Section A.3.1). This analysis identified 23 COPCs that warranted a more detailed analysis. Of the 23 COPCs, total PCBs and BEHP were the two chemicals with more frequent exceedances of the SQS and CSL than any other chemicals in the sediments.

CSL exceedances of the highest priority COPCs (based on frequency of detection and standards/guideline exceedance) were generally co-located with CSL exceedances of

[102] PCBs and BEHP were also evaluated individually.

[103] TBT is also a benthic COPC, but was assessed using a tissue approach rather than a bulk sediment guideline.

either BEHP or total PCBs. Using a GIS analysis of multiple chemicals, multiple criterion exceedances were identified at the following areas: south of Harbor Island (RM 0.1[104]), RM 0.3 - 0.6 (east side), Slip 3 and the west side opposite Slip 3 (RM 1.9 - 2.2), most of the east side between Slips 4 and 6, and upstream of the turning basin (RM 4.8 - 5.5).

Additional fieldwork in Phase 2 is recommended to reduce the uncertainties identified in the benthic invertebrate assessment, and risk to benthic invertebrates will be further evaluated in the Phase 2 ERA.

A.7.1.3.2 Benthic invertebrates and tissue chemistry

Crab

A summary of the risk characterization for crab is presented in Table A-7-14. Risk to crabs from sediment-associated COPCs in the LDW appeared to be relatively low based on the limited data available on exposures and effects, with the possible exception of arsenic, which had a NOEC-based HQ of 10. However, this HQ was based on a NOEC only, because no studies were found in the literature associating a tissue arsenic concentration with an adverse effect in crabs or related species. Therefore, this HQ is highly uncertain.

Table A-7-14. Summary of risk characterization results for chemicals measured in crab tissue

COPC	Highest NOEC HQ	No. of TRV Studies	Uncertainty in TRV	Exposure Data	
				Edible Meat (n)	Hepatopancreas (n)
Arsenic	**10**	1	high [a]	2	1
Cadmium	0.008	1	high [b]	2	1
Chromium	0.15	1	medium	2	1
Copper	0.59	1	high [c]	2	1
Lead	na	0	na	2	1
Mercury	0.68	2	medium	5	1
Nickel	na	0	na	2	1
Silver	na	0	na	2	1
Zinc	0.45	1	high [d]	2	1
PAHs	na	0	na	2	1
Phthalates	na	0	na	2	1
SVOCs	na	0	na	2	1
TBT	0.65	1	medium	5	1
PCBs	0.02	1	medium	5	1
VOCs	na	0	na	2	1

Note: HQs greater than 1.0 are noted in **bold type**

na – not applicable

[104] As measured from the southern tip of Harbor Island.

[a] Only one study with shrimp

[b] Only one study, which measured cadmium in muscle only

[c] Only one study, which measured copper in crayfish claws

[d] Only one study, which measured zinc in crayfish

Based on the arsenic NOEC-based HQ of 10, and despite the uncertainties associated with tissue data and TRVs (but primarily due to TRVs) (Table A-7-14), collection of additional crab tissue data is recommended for Phase 2, and risk to crab will be further evaluated in the Phase 2 ERA.

TBT Assessment

A range of HQs was calculated using either measured maximum tissue concentrations, or tissue concentrations estimated using minimum, median, or maximum sediment concentrations. Due to the potentially high HQ (up to 5.3 using the maximum sediment concentration to estimate the tissue concentration), TBT is recommended for further evaluation in the Phase 2 ERA for risks to benthic invertebrates.

The Phase 2 ERA will also further address the TBT TRV selected to calculate these HQs because some of the sublethal effects are specific to a small group of species, specifically induction of imposex or intersex in meso- and neogastropod snails. Six gastropod orders have been reported in the LDW (Table A-2-2), including Meso- and Neogastropoda. Among the sensitive snail species tested for imposex, only periwinkle and dogwhelk species have been found in the Pacific Northwest (Section A.7.1.2.2), although they have not been observed in the few benthic invertebrate surveys that have been conducted in the LDW. However, other meso- and neogastropod species have been found in the LDW (Table A-2-2). Therefore, collection of additional data to support the TBT analysis for benthic invertebrates is recommended for Phase 2.

A.7.2 Risk Characterization for Fish

This section presents an estimation of risk to fish species in the LDW by calculating HQs for each of the three fish ROCs using either tissue residue or dietary exposure approaches described in Section A.4.1 along with TRVs presented in Section A.4.2. Following the risk estimation, a detailed evaluation of uncertainty associated with these calculations is presented. Finally, this section presents a risk conclusion that integrates HQ results with associated uncertainty to summarize the results of the Phase 1 risk assessment based on available data, and provides input to the data gaps process. ROC/COPC pairs to be evaluated in the Phase 2 ERA will be determined in the Phase 2 problem formulation following a process described in the Phase 2 work plan.

A.7.2.1 Risk estimation

This section presents the HQ calculations for fish ROC/COPC pairs. Dietary and tissue residue based HQs are presented in Table A-7-15. Results for each ROC are described in the sections below.

Table A-7-15. HQs for fish ROC/COPC pairs

		Dietary HQs					
		Arsenic		Copper		PAHs	
		NOEC	LOEC	NOEC	LOEC	NOEC	LOEC
Juvenile chinook	Survival	na	na	0.23	na	0.03	na
	Growth	0.41	0.27	0.24	0.24	**1.7**	0.17
	Reproduction	ne	ne	ne	ne	ne	ne
Bull trout	Survival	na	na	0.01	na	ne	ne
	Growth	0.29	0.19	0.01	0.01	ne	ne
	Reproduction	na	na	na	na	na	na
English sole	Survival	na	na	0.17	na	0.0006	na
	Growth	**1.6**	**1.1**	**15**	**7.6**	0.07	0.03
	Reproduction	na	na	na	na	na	na

		Critical Tissue Residue HQs							
		Mercury		TBT		DDTs		PCBs	
		NOEC	LOEC	NOEC	LOEC	NOEC	LOEC	NOEC	LOEC
Juvenile chinook	Survival (composite)	0.003	na	**1.1**	0.11	0.02	0.01	0.001[a]	0.0003
	Survival (individual)	na	na	na	na	0.01	0.006	0.002[a]	0.0004
	Growth (composite)	0.01	0.009	na	na	0.005	na	0.002	0.001
	Growth (individual)	na	na	na	na	0.002	na	0.004	0.002
	Reproduction	ne	ne	ne	ne	ne	ne	ne	ne
Bull trout	Survival	**2.2**	0.94	**1.1**	0.11	0.06	0.04	0.30	0.18
	Growth	0.55	0.34	na	na	0.01	na	**8.2**	**2.1**
	Reproduction	**2.1**	0.21	1.0	0.10	0.37	0.04	**4.2**	0.87
English sole	Survival	0.38	0.16	0.11	0.01	0.01	0.002	0.09	0.05
	Growth	0.10	0.06	na	na	0.0009	na	**2.4**	0.62
	Reproduction	0.36	0.04	0.11	0.01	0.02	0.002	**1.2**	0.25

Note: HQs greater than 1.0 are noted in **bold type**

ne – Not evaluated in the EEA (considered to pose negligible risk based on analyses in the problem formulation)

na – No HQ available because of lack of relevant toxicity data

[a] Value also represents HQ for survival following immunological challenge

A.7.2.1.1 Juvenile chinook salmon

COPCs identified for juvenile chinook salmon in the problem formulation were arsenic, copper, PAHs, mercury, TBT, DDT, and PCBs. NO LOEC-based HQs were greater than 1. Thus, exposure greater than concentrations associated with observed effects was not estimated for any of the COPCs based on available data. For NOEC-based HQs, only two of seven COPCs' HQs were greater than 1. The NOEC-based HQ for PAHs was 1.7 for the growth endpoint and the NOEC-based HQ for TBT was 1.1 for the survival endpoint. Because these HQs were not associated with TRVs for which adverse effects were observed, the potential for effects is less certain.

A.7.2.1.2 Bull trout

Biomagnifying COPCs

In this phase 1 ERA, bull trout were selected to represent risks from biomagnifying COPCs to all piscivorous fish in the LDW (see Section A.2.3.2.2). Biomagnifying COPCs for bull trout included mercury, DDTs, and PCBs. The only LOEC-based HQ exceeding 1 was for PCBs and growth (2.1). Four NOEC-based HQs exceeded 1 for biomagnifying compounds, although, as discussed above, the interpretation of these exceedances is more uncertain. For mercury, NOEC-based HQs were 2.2 and 2.1 for survival and reproductive endpoints, respectively. For PCBs, the NOEC-based HQs for growth and reproduction were greater than 1 (8.2 and 4.3, respectively). Because all available and appropriate fish tissue data were used to model bull trout exposure, the outcome of the modeling effort would have produced similar exposure estimates for any pelagic piscivore in the LDW. As such, all pelagic piscivores should be represented by this assessment to the extent possible with the available data. However, some uncertainty is associated with the assumption that bull trout exposure is representative of other benthic piscivores. The collection and analysis of additional piscivorous fish tissue will be discussed in the data gaps memorandum.

Nonbiomagnifying COPCs

Risks from non-biomagnifying COPCs were evaluated for bull trout because of its endangered status and differing pathway (i.e., piscivorous diet; see Section A.2.3.2.2). Nonbiomagnifying COPCs for bull trout included arsenic, copper, and TBT. No LOEC-based HQs exceeded 1 for non-biomagnifying COPCs based on existing data (Table A-7-15). Only TBT had a NOEC-based HQ exceeding 1 (1.1 for the survival endpoint and 1.0 for the reproduction endpoint). Because the TBT HQ was not associated with a TRV for which adverse effects were observed, the potential for effects is uncertain.

A.7.2.1.3 English sole

COPCs for English sole included arsenic, copper, PAHs, mercury, TBT, DDTs, and PCBs. For arsenic and copper, both the LOEC- and NOEC-based HQs exceeded 1 for the growth endpoint (Table A-7-15). NOEC- and LOEC-based HQs for copper of 15 and 7.6, respectively, were calculated for the growth endpoint. The reproduction

endpoint was not evaluated for arsenic, copper, or PAHs because sufficient toxicological data were not available. For PCBs, the NOEC-based HQs for the survival and reproduction endpoints were 2.4 and 1.2, respectively. No LOEC-based HQs for PCBs exceeded 1. NOEC- and LOEC-based HQs for mercury, TBT, and DDT did not exceed 1. Therefore, based on existing data, the only effects-based TRVs exceeded were for dietary arsenic and copper exposure. Exceedance of the PCB HQ was based on a no-effects TRV, so the potential for effects is less certain.

A.7.2.2 Uncertainty assessment

This section presents a discussion of uncertainty associated with the problem formulation and the exposure and effects assessments for fish ROCs. An uncertainty is considered to have the potential to impact risk conclusions if alternative calculations or relatively small changes in exposure data could result in a change of an HQ from less than 1 to greater than 1, or vice versa.

At the end of this section, key areas of uncertainty are summarized and qualitatively ranked as low, medium, or high according to their level of uncertainty, potential to impact risk conclusions, and overall feasibility of reducing uncertainty either through literature-based analysis or field studies.

A.7.2.2.1 Problem formulation

Primary uncertainties in the problem formulation for fish include ROC selection, assessment endpoints, pathways, and the COPC screen. These uncertainties are discussed in the following sections.

ROC Selection

The LDW supports a varied fish community with freshwater, marine, and anadromous species present from 24 different families. Due to this variability, there is some uncertainty as to whether the three selected ROCs (juvenile chinook salmon, bull trout, and English sole; see Section A.2.3.2) are sufficient to represent a worst-case scenario for combined sensitivity and exposure for other fish species present in the LDW. Uncertainties associated with each ROC are discussed below.

Juvenile chinook salmon

For purposes of this assessment, wild juvenile chinook salmon represented all migratory juvenile salmonids in the LDW. Uncertainty exists as to whether juvenile chinook salmon have the greatest residence time of all juvenile salmonids in the LDW. Chum salmon are also suspected to rear in the LDW for an extended period. If juvenile chum salmon have greater residence in the LDW than juvenile chinook salmon, they could have higher tissue burdens, and predicted risk for some COPCs could be underestimated. Tissue residue data were not available for juvenile chum salmon from the LDW, so a direct comparison of exposure could not be conducted. Warner and Fritz (1995) observed that juvenile chum salmon are present in large numbers in the LDW from April through June. Continuous and even increases over time in size of

chum sampled during this period suggest they may rear extensively in the LDW (Warner and Fritz 1995). In this study, chum populations were present for a shorter time in the estuary than chinook populations (early April - July, and late March - September, respectively). Neither individual juvenile chum salmon nor individual chinook salmon have been tracked through the LDW, so residence time is uncertain for these species. Juvenile chinook salmon are generally regarded as the most estuarine dependent juvenile salmonid (Groot and Margolis 1991). Assuming this is true for the LDW, their exposure to sediment-associated chemicals is likely equal to or greater than other juvenile salmonids.

There is uncertainty as to whether wild yearling juvenile chinook salmon are present in the LDW. Hatchery yearlings ranging in size from approximately 120 to 180 mm have been captured in the LDW. These fish were likely hatchery yearlings released for the blackmouth fishery or stranded hatchery fish that overwinter in the Howard Hansen reservoir (both of which enter the LDW as yearlings) (Warner and Fritz 1995). Warner and Fritz (1995) state that their catch data supports the hypothesis that no wild yearling chinook are produced in the LDW and that yearling hatchery chinook appeared to move through the LDW quickly[105] (based on frequent seining at multiple locations in and above the LDW). However, unclipped juvenile chinook salmon up to 176 mm have also been captured in the LDW (Shannon 2001) suggesting that, contrary to Warner and Fritz (1995), wild yearling chinook may be present in the LDW. Alternatively, these fish could be unclipped hatchery fish. Larger juvenile chinook are generally associated with deeper water and have dietary preferences that may include juvenile fish (Healy 1991). Yearling juvenile chinook are expected to have shorter residence in the LDW than subyearling juvenile chinook (Healy 1991; Warner and Fritz 1995), thus they should have less opportunity for exposure. In addition, because risks to piscivorous fish (i.e., bull trout; see below) were assessed, they are likely to be representative of risk to piscivorous yearling juvenile chinook salmon not covered by the juvenile chinook salmon ROC. As such, juvenile chinook salmon are likely an appropriate ROC to represent migratory juvenile salmonids.

Bull trout

Bull trout were selected as an ROC to represent all piscivorous fish in the LDW for biomagnifying chemicals, including fish that spawn in the LDW. No whole-body tissue residue data were available for any species of piscivorous fish collected from the LDW. Therefore, all exposure analyses for the bull trout ROC were based on modeled data (see Section A.4.1.1). Because all available and appropriate fish tissue data were used to model bull trout exposure, and model assumptions were generic rather than specific to bull trout (e.g., predator prey factors), the outcome of this modeling effort would have produced similar exposure estimates for any pelagic piscivore in the LDW. In the effects assessment, for chemicals that biomagnify (i.e., mercury, DDTs, &

[105] No estimate of residence time is provided, though based on this comment, residence is likely to be considerably shorter than that of subyearling juvenile chinook salmon.

PCBs), TRVs for bull trout were selected from studies of all families of fish, rather than just the family Salmonidae. As such, all pelagic piscivores should be represented by this assessment to the extent possible with the available data. Some uncertainty is associated, however, with the assumption that pelagic piscivores appropriately represent benthic piscivores. The magnitude of this uncertainty for a given species depends on its relative trophic status and extent of direct sediment exposure. The collection and analysis of additional piscivorous fish tissue will be discussed in the data gaps memorandum.

English sole

The English sole ROC was evaluated to represent all fish in the LDW not specifically covered by juvenile chinook salmon (anadromous juvenile salmonids) or bull trout (piscivorous fish). By using the approach outlined in Section A.7.2.3, HQs calculated for English sole did not necessarily reflect risk specific to any particular fish (or English sole), but provided an estimate of risk to fish in the LDW that may be both sensitive and highly exposed. In the English sole assessment, tissue concentrations in this highly exposed fish (due to direct sediment contact and benthivorous diet) were compared to the lowest available TRVs for any fish species. Because it is unlikely that the most exposed fish is also the most sensitive, risks estimated for English sole in this ERA should provide a conservative estimate for risks to fish represented by this ROC in the LDW.

In summary, there is some uncertainty associated with selection of juvenile chinook salmon, bull trout, and English sole as ROCs representative of the overall fish community in the LDW. Based on available data, selection of additional or different ROCs (with the possible exception of a benthic piscivore) would not likely change the overall results of the Phase 1 ERA due to conservative assumptions used throughout the assessment. Collection of additional tissue residue data for fish species, including an upper-trophic-level benthic piscivore is recommended, and will be discussed in the data gaps memorandum.

Assessment Endpoints

Endpoints for fish ROCs included direct effects on survival, growth and reproduction (only bull trout and English sole were evaluated for reproduction endpoint). Effects on survival due to reduced immunocompetence for juvenile chinook salmon from exposure to PCBs and PAHs, and effects associated with cancerous lesions for English sole from PAH exposure, were also included. However, several other biochemical, histological and or physiological alterations or manifestations of stress that have been shown as a response to chemical exposure by fish were not included in this ERA. These biomarkers of effect at the organismal and suborganismal level may provide an early warning of adverse effects on fish at the individual and population levels (Payne et al. 1987; Schmitt and Dethloff 2000).

Biomarkers currently being investigated by the scientific community as potential indications of adverse effects include cytochrome P450-dependant monooxygenases

induction (e.g., CYP1a [van der Weiden et al. 1994]), DNA changes (e.g., DNA-xenobiotic adduct formation [(Rice et al. 2000]), and sub-organismal immune dysfunction (e.g., antibody formation [Arkoosh et al. 1991]). Frequently, biomarkers are induced at lower chemical concentrations than responses resulting in direct effects on survival, growth, and reproduction, suggesting that some change may occur at these lower doses. From an individual or population level, however, the overall significance of these changes is unknown. Thus, risk estimates were limited to survival, growth, and reproduction in this Phase 1 ERA.

Exposure Pathways

Three exposure pathways associated with sediment contamination and fish ROCs were designated as complete, but of unknown significance (Figure A-2-2). These pathways included juvenile chinook salmon drift organism ingestion, and English sole direct sediment contact and sediment ingestion.

In addition to benthic invertebrates, juvenile chinook salmon in the LDW rely extensively on drift organisms such as wasps and ants (Cordell et al. 1997, 1999). It is uncertain, but unlikely, that juvenile chinook salmon are significantly exposed to sediment-associated COPCs from consumption of these organisms. Several flying insects such as dipteran flies have larval stages that live in the sediment. It is possible that adult life stages of these insects may have chemical tissue residues associated with larval sediment exposure, and that this exposure may be passed on to juvenile chinook salmon. Dipterans are a major component of juvenile chinook salmon diet in other Pacific Northwest estuaries, according to Cordell et al. (2001). However, exclusion of this pathway from the exposure analysis is not likely to significantly underestimate risk predictions based on consumption of benthic invertebrates. Benthic invertebrates are likely to have greater exposure to sediment-associated chemicals than would adult terrestrial insects.

For English sole, direct sediment contact was designated as a complete pathway of unknown significance. English sole live in close contact with sediments and likely are exposed to sediment-associated chemicals. However, no effects data were available to estimate potential impacts from direct contact. Therefore, the magnitude of this uncertainty is unknown.

For English sole, sediment ingestion was a complete pathway of unknown significance. English sole rely extensively on benthic invertebrates as prey (e.g., marine worms and amphipods). It is likely that by consuming these animals some incidental ingestion of sediment occurs. No information on stomach content of English sole from the LDW was available, and no information on sediment consumption by English sole was identified in any study. To account for this uncertainty in the exposure assessment, incidental sediment ingestion was assumed to account for 10% of the overall English sole diet for COPCs analyzed via a dietary pathway (metals and PAHs) (Section A.4.1.2). Selection of 10% was based purely on best professional judgment; no stomach content data or other data were available to verify this

assumption. This uncertainty is addressed further in section A.7.2.2.2 by calculating exposure assuming different sediment ingestion at different dietary proportions. For bioaccumulating chemicals analyzed using a tissue residue approach, this pathway was incorporated into tissue residues. Thus, for bioaccumulating chemicals, this uncertainty does not affect risk conclusions.

In summary, three sediment exposure pathways for fish ROCs were classified as complete, but of unknown significance. However, as discussed above, juvenile chinook salmon ingestion of drift organisms and English sole ingestion of sediment likely constitute relatively small proportions of their overall exposure, and inclusion of these pathways is not likely to impact risk conclusions. The magnitude of uncertainty is unknown for nonbiomagnifying chemicals in English sole as a result of direct sediment contact.

Also, PAHs were not evaluated as a COPC for bull trout based on an incomplete pathway due to their piscivorous diet. A worst-case scenario estimate was made to evaluate potential exposure of bull trout to PAHs if benthic invertebrates were consumed. Potential bull trout PAH exposure was calculated assuming 100% consumption of amphipods from the most contaminated site in the LDW. Amphipod data were modeled by applying a BSAF calculated from Kellogg Island synoptic sediment and amphipod data to the highest total PAH sediment concentration from the LDW using the following equation:

$$BSAF = \frac{\text{Biota (µg/g lipid)}}{\text{Sediment (mg/kg OC)}} = \frac{4.74}{226} = 0.021 \qquad \textit{Equation 7-1}$$

Data used to calculate the BSAF are presented in Table A-7-16. The estimated biota (amphipod) tissue concentration was calculated from the BSAF and the LDW maximum total PAH sediment concentration of 11,594 mg/kg OC to obtain a dietary exposure value for comparison to the TRV. The estimated LDW amphipod tissue concentration was calculated as follows:

$$\text{Biota}(\mu g/g \text{ lipid}) = BSAF \times \text{sediment}(mg/kg\ OC) = 0.021 \times 11{,}594 = 244\ \mu g/g \text{ lipid}$$

Assuming a 5% lipid content (approximate content in West Marginal Way amphipod samples), the weight-based concentration was calculated as follows:

$$\text{Biota}(\mu g/g) = 244\ \mu g/g \text{ lipid} \times 0.05 = 12\ \mu g/g \text{ ww}$$

The TRV was assumed to be a dry weight value, so the above LDW estimated biota dietary concentration of 12 µg/g was converted to a dry-weight value assuming a solids content of 20%. The resulting estimated maximum dietary amphipod concentration on a dry-weight basis was 61 µg/g. This value was lower than the NOEC of 100 µg/g -diet (dw) for growth of rainbow trout (Hart and Heddle 1991), indicating that risk to bull trout from ingestion of amphipods in the LDW is unlikely. This analysis is uncertain because of the number of assumptions involved and the lack

of direct exposure data, e.g., stomach content data. Due to the low likelihood of a piscivorous fish feeding only on benthic invertebrates from the most contaminated spot, risk to piscivorous fish from exposure to PAHs is low and not likely to affect risk conclusions.

Table A-7-16. Amphipod Total PAH BSAF

Location	Amphipod Total PAH Concentration (µg/g lipid)	Sediment Total PAH Concentration (mg/kg OC)	BSAF
Kellogg Island	Not detected[a]	45.5	Not calculated
West Marginal Way	4.74[b]	226	0.021

[a] Method detection limits (MDLs) ranged from 16 to 64 µg/kg for individual PAHs; the sum of MDLs was 584 µg/kg (approximately 11.7 µg/g lipid).

[b] Only one of two samples had detectable PAHs; only pyrene and fluoranthene were detected.

COPC Screen

Some chemicals have been detected in LDW sediment and in LDW tissue for which no toxicological data exist. Of the chemicals detected in LDW sediment at a frequency greater than 5% (40 chemicals), four chemicals were not evaluated for potential adverse effects to fish: 4-methylphenol, benzoic acid, butyl benzyl phthalate, and dibenzofuran. Whole-body fish tissue data were available for these chemicals in three composite samples of shiner surfperch collected from the vicinity of Kellogg Island in the LDW. Amphipod tissue residue data were also available for these chemicals from four composite samples collected in the vicinity of Kellogg Island. All tissue residues for these five chemicals were below analytical detection limits except benzoic acid, which was detected in all three shiner surfperch samples. However, no toxicological data were identified that could be used to screen fish for any of these chemicals. Because toxicological data for these chemicals were not available, risk associated with these chemicals is unknown.

Toxicological data are available for some chemicals that have not been analyzed in LDW fish tissue. The potential for these chemicals to be present in LDW sediments and tissues at concentrations potentially toxic to fish has not been assessed. Chemicals for which toxicological data are available, but no tissue residue data exist, will be acknowledged in the data gaps memorandum. If new data are collected, these chemicals will be further analyzed for risk to fish in the Phase 2 ERA.

A.7.2.2.2 Exposure assessment

Uncertainty associated with tissue data, dietary composition, site use, and potential future restoration projects are addressed in this section. Each of these topics is summarized in the following bullets, and discussed in more detail in the subsection below.

- Tissue data uncertainty including the limited number of samples available, the limited area over which samples were collected, modeled data, use of juvenile

chinook salmon stomach contents data, and differences in calculating total PAH and total PCBs between studies

- Dietary composition uncertainty including lack of data on dietary composition for the ROCs, assumptions used in modeling dietary exposure, and dietary exposure from sources outside the LDW
- Site use uncertainty including limited site use data for fish ROCs and assumptions regarding site use
- Potential restoration sites including uncertainty due to changes in habitat in the LDW

Tissue Data

Tissue data used in this Phase 1 ERA were collected for various purposes from a variety of programs. Because these data were not explicitly generated for the purpose of conducting an ERA, there are several sources of uncertainty associated with their use. A small number of tissue samples were available for amphipods, crab, perch, and English sole. Additionally, most of these samples were collected from the lower section of the LDW. Because of the limited data available, it was necessary to estimate some data (e.g., bull trout whole-body tissue data estimated from PPFs and available fish tissue data). This section discusses the uncertainty associated with use of the available tissue data and potential effects of these uncertainties on risk conclusions for the affected ROC/COPC pairs.

This section discusses these uncertainties under the following headings:

- Limited data availability
- Modeled data
- Use of juvenile chinook salmon stomach contents data
- Calculation of total PAHs and PCBs.

Limited data availability

This section addresses uncertainty associated with the quantity and representativeness of the available data. Areas of uncertainty discussed in this section include the small number of available tissue samples for the ROCs and their prey, the limited area over which tissue samples were collected, additional juvenile chinook salmon tissue data not used in the ERA, and the age of organisms sampled relative to age of organisms consumed as prey by the ROCs.

A small number of tissue samples were collected, particularly for amphipods, mussels, crab, perch, and sole; numbers of composite samples ranged from two to four. Although the number of samples analyzed was low, these samples were composites of three individuals for crab, 22 for mussels, 10 for perch, 20 for English sole, and about 2,000 for amphipods. Composite samples incorporate variability in individual organisms, thus reducing overall uncertainty to some extent. However, due to the

limited tissue residue data available, all conclusions based on these data are somewhat uncertain. Based on this limitation, risk has been interpreted conservatively in the Phase 1 ERA. Additional tissue data will be collected for use in the Phase 2 ERA.

No juvenile chinook salmon TBT tissue data were available. Lack of juvenile chinook salmon data increases the uncertainty of risk conclusions for TBT. However, TBT data were available for perch, which are likely exposed to a greater extent than juvenile chinook salmon due to their greater residence time and similar diet.

Crab, shiner surfperch, and English sole whole-body tissue samples were collected only in the lower portion of the LDW below RM 1.5. There is uncertainty regarding movement of these species within LDW and whether available tissue residue data serve as an appropriate surrogate for fish from other areas of the LDW. Shiner surfperch are generally found throughout the LDW (Miller 1977a), but information was not available as to the size of their home range. English sole are most abundant in the lower portion of the river (based on otter trawl data), although they are seasonally present throughout the LDW (Miller 1977a). Due to these uncertainties, feasibility and utility of collecting additional fish and fish prey tissue data will be further addressed in the data gaps memorandum.

All juvenile chinook salmon tissue data were collected from the lower waterway in the vicinity of Kellogg Island and Slip 4. Because juvenile chinook salmon captured in the lower section of the LDW have migrated through the upper sections of the LDW, these fish likely represent exposure averaged over the entire LDW. Most of the available tissue data were for hatchery released juvenile chinook salmon that were exposed to low levels of COPCs, such as PCBs, at the hatchery through their feed, and in the Green River above the LDW. Thus, the uncertainty associated with tissue residue in these fish may slightly overestimate chemical exposure from LDW sediments. Because no juvenile chinook salmon HQs calculated using tissue residues were greater than 1, uncertainty from this source does not likely affect the risk conclusions. [106]The age class of shiner surfperch for which whole-body data were available (adults) may not represent chemical concentrations in juvenile fish consumed by bull trout (and other piscivores represented by this ROC). These data were used to estimate bull trout exposure to arsenic, cadmium, chromium, copper, lead, silver, zinc, TBT, and mercury in the problem formulation. Because older fish are exposed over a longer period of time, they are likely to have higher tissue concentrations of some chemicals (e.g., mercury) than juvenile fish. Therefore, based on this uncertainty, risk to bull trout from these COPCs may be overestimated. Of these chemicals HQs were greater than 1 only for mercury and TBT (maximum HQs of 2.2 and 2.0, respectively). The feasibility and utility of collecting piscivorous fish tissue data, and tissue of their prey will be addressed in the data gaps memorandum.

[106] Note, however, that relatively little is known regarding specific site usage in the LDW. If some juvenile salmonids spend a disproportionate amount of time in an area with higher COPC concentrations, then the available data could underestimate their exposure.

The age class of Dungeness crab (assumed to be adult) for which whole-body data were estimated may not represent chemical concentrations in crab consumed by English sole (juveniles). These data were used to estimate English sole exposure to copper and arsenic. Because older crab are exposed over a longer period of time, they are likely to have higher tissue concentrations of some chemicals (e.g., mercury) than juvenile crab. Therefore, risk to English sole from these COPCs may have been overestimated. The feasibility and utility of collecting additional English sole prey tissue data will be further discussed in the data gaps memorandum.

Modeled data

This section addresses uncertainty associated with use of the available data to estimate concentrations in different tissues. Areas of uncertainty discussed in this section include:

- use of PSAMP English sole data and fillet data to predict English sole whole body data
- use of PPFs to predict piscivorous fish whole body data
- use of amphipod data collected from the vicinity of Kellogg Island to predict amphipod metals tissue data from throughout the LDW
- use of crab hepatopancreas and edible meat data to predict crab whole body data

English Sole Data. There is uncertainty associated with the use of whole-body residue data estimated from tissue for English sole collected during a PSAMP sampling event on April 14, 1997. During this event, 60 adult (greater than 9") English sole were collected and sorted into three groups of 20 fish each (composite mean lengths were 247, 275, and 333 mm, respectively for small, medium, and large fish). These fish were then subsampled for PSAMP purposes. Specifically, 5 to 10 g of muscle tissue was dissected from the fish for chemical analysis (fish appeared to weigh about 140 to 190 g each). In addition, some fish were eviscerated to collect liver histopathology data. Fish were then packaged and sent to John Strand for the King County WQA. Strand (2002) stated that the fish he received included bones, skins, heads, tails and viscera (less gonads), although livers were only present in some fish (percent is not known).

Lack of livers and portions of other tissue in English sole samples results in uncertainty associated with use of these data to approximate whole-body concentrations. For example, PCBs tend to concentrate in the liver due to their affinity for lipids and the high lipid concentrations in livers. Therefore, whole-body concentrations for samples lacking liver tissue may underestimate a true whole body concentration. Because liver and fillet data were available from 1992 and 1995 PSAMP sampling events and fillet data from the 1997 samples, these data were used to approximate whole body concentrations. Over all years, fillet PCB concentrations ranged from 0.079 to 0.36 µg/g ww. In 1992 and 1995, PCB concentrations in six

composite liver samples ranged from 3.1 to 7.1 µg/g ww. Assuming the liver constitutes 10% of the English sole body (based on best professional judgment), by wet weight, a rough calculation was made to assess the potential effect on the estimated whole body concentrations if the tissue these data were based on did not include the liver. Using the highest available whole-body PCB concentration in English sole (2.3 µg/kg ww) and the highest liver PCB concentration (7.1 µg/kg ww) and assuming the liver constituted 10% of the fish, the maximum estimated whole-body concentration could increase from 2.3 to 2.8 µg/kg ww, an increase of approximately 21% based on the whole-body English sole samples discussed above. The maximum HQ (the growth NOEC HQ) would increase from 2.4 to 2.9. Therefore, this increase would not have changed the risk conclusions presented for English sole (Section A.7.2.3).

To expand the English sole tissue residue dataset, stakeholders have questioned whether fillet data could be used to estimate whole-body tissue residue data by estimating a fillet-to-whole-body ratio using existing data. Typically, when studies attempt to estimate the whole-body-to-fillet concentration ratio, care is taken to keep the whole-body sample as intact as possible. However, as discussed above, the available whole-body data were based on samples that may have been missing liver or other tissue. Therefore, estimates of a fillet-to-whole-body ratio are highly uncertain.

English sole exposure to DDTs was based on tissue residues reported for nine English sole fillet composite samples collected throughout the LDW. Use of fillet data to represent whole-body tissue residues likely underestimates exposure for chemicals such as DDTs that tend to accumulate in fatty tissue. However, the maximum DDT HQ for English sole was 0.023 for the reproduction endpoint. Thus, whole-body tissue residues of DDTs would have to be 43 times higher than fillet samples to attain an HQ of 1. While it is possible high whole-body-to-fillet ratios could occur in English sole, it is unlikely that they would be high enough to result in HQs greater than 1. Therefore, this uncertainty is unlikely to significantly affect the risk conclusion for English sole and DDTs.

Use of PPFs. No tissue residue data were available for bull trout or any other piscivorous fish from the LDW. As previously discussed, tissue burdens in bull trout were estimated based on available English sole, shiner surfperch, and juvenile chinook salmon data and a PPF for biomagnifying chemicals. The PPFs were derived from mercury (EPA 1997c) and PCB studies (Metcalfe and Metcalfe 1997). Because of the somewhat similar structure of PCBs and DDTs (i.e., both are hydrophobic organochlorines), the PPFs for these chemicals were assumed to be similar. The PCB PPF of 3.5 was based on average tissue residues in alewife and lake trout in northern Lake Ontario (Metcalfe and Metcalfe 1997). Tissue residues were variable in both species and a maximum PPF of approximately 9 could be calculated based on these data. Actual PPFs are likely to vary between species and are influenced by factors such as lipid concentrations and relative trophic position (Metcalfe and Metcalfe 1997). The PPF for mercury is also potentially highly variable (EPA 1997c).

Effects on HQs associated with using maximum PPFs (i.e., 10 for mercury, or double the average PPF; and 9 for PCBs, the maximum PPF for lake trout based on Metcalfe and Metcalfe [1997]) are shown in Table A-7-17. Use of these maximum PPFs results in elevation of the survival LOEC-based HQ for mercury to greater than 1, and the growth and reproduction LOEC-based HQ for PCBs to greater than 1. The maximum NOEC-based HQ for DDT was still less than 1, so increasing the PPF from 3.5 to 9 would not change the risk conclusion for bull trout/DDT. The feasibility and utility of collecting additional piscivorous fish tissue for analysis will be addressed in the data gaps memorandum.

Table A-7-17. Effects of using different PPFs[a] on HQs for bull trout

Endpoint		Critical Tissue Residue HQs					
		Mercury		PCBs		DDT	
		NOEC	LOEC	NOEC	LOEC	NOEC	LOEC
Survival	HQ (original)	**2.2**	0.94	0.30	0.02	0.06	0.04
	HQ (maximum)	**4.4**	**1.9**	0.77	**1.5**	0.15	0.09
Growth	HQ (original)	0.55	0.34	8.2	**2.1**	0.01	na
	HQ (maximum)	**1.1**	0.67	21	**5.5**	0.04	na
Reproduction	HQ (original)	**2.1**	0.21	**4.2**	0.87	0.37	0.04
	HQ (maximum)	**4.2**	0.42	11	**2.2**	0.93	0.09

HQs greater than 1.0 are noted in **bold type**.

[a] Increased mercury PPF from 5 to 10, and increased PCB PPF from 3.5 to 9. DDT PPF was assumed to be equivalent to the PCB PPF.

na – Not available

Amphipod Data. Amphipod tissue residue data were used to model juvenile chinook salmon exposure to arsenic, cadmium, chromium, copper, lead, silver, and zinc; and English sole exposure to arsenic, cadmium, chromium, copper, lead, silver, zinc and PAHs.[107] As previously discussed, amphipods were collected near the west side of Kellogg Island where sediment contamination was generally low to moderate, and thus, these tissue concentrations may not be reflective of worst-case exposure in the LDW. To evaluate the degree to which these amphipod tissue residues would approximate SWA dietary exposure through amphipod consumption throughout the LDW, chemical concentrations in sediments collected synoptically with amphipod tissue were compared with the SWA sediment concentrations in the entire LDW (Table A-7-18). This comparison is relevant if juvenile chinook salmon and English sole forage throughout the LDW in a non-preferential manner, and if these COPCs accumulate in amphipod tissue proportionally throughout the LDW.

[107] Note that all except arsenic, copper, and PAHs were screened out in the problem formulation.

Table A-7-18. Comparison of LDW SWA sediment concentrations with sediments collected synoptically with amphipods

Chemical	West Marginal Way Sediment (mg/kg dw)	Kellogg Island Sediment (mg/kg dw)	LDW SWA Sediment (mg/kg dw)
Arsenic	12	5.7	12
Cadmium	0.16	0.28	0.44
Copper	65	20	58
Chromium	19	16	29
Lead	108	17	48
Silver	0.22	0.14	0.41
Zinc	117	45	120
TPAH[a]	29	1.0	4.4

[a] Non-detected PAHs were not included in calculation of total PAHs

For arsenic, copper, lead, and TPAH, the SWA concentration was between those observed at the Kellogg Island and West Marginal Way sampling locations. Therefore, average amphipod COPC concentrations (corresponding to SWA) for four of the eight chemicals should be within the range of COPCs measured in amphipods collected near Kellogg Island. Sediment concentrations of cadmium, chromium, silver, and zinc collected synoptically with amphipods near Kellogg Island were lower than the LDW SWAs for these chemicals. Therefore, average amphipod concentrations for these metals may be higher than those measured near Kellogg Island. Based on the above analysis, the amphipod tissue data for the other chemicals (i.e., arsenic, copper, lead, and TPAHs) should be reflective of average juvenile chinook salmon and English sole exposure in the LDW, assuming equal usage of all habitat.

Use of juvenile chinook salmon stomach contents data

Juvenile chinook salmon stomach content data were used to estimate juvenile chinook salmon exposure to PAHs, thus incorporating additional sources of exposure beyond that received from epibenthic invertebrates (e.g., amphipods). PAH concentrations in stomach contents of juvenile chinook salmon collected from the LDW were much higher than those suggested by PAH concentrations in benthic invertebrates collected from the LDW (e.g., amphipods, crabs) suggesting an additional source of dietary PAHs may exist for juvenile chinook salmon. A possible explanation for the greater PAH concentrations is consumption of contaminated drift insects (e.g., wasps, ants exposed to PAHs in the water surface microlayer) or exposure to concentrated substances such as creosote, tar balls, oil, or grease. Alternatively, the PAH concentrations of ingested benthic invertebrates may not be well represented by the modeled amphipod data. Thus, the available stomach content data may over- or underestimate risk from sediment-associated PAHs.

Calculation of total PAHs and PCBs

Total PCB data for juvenile chinook salmon and English sole tissue were calculated based on the sum of PCB homologues (tri- through deca-) and the sum of Aroclors, respectively. The PCB effects data, however, were based on individual Aroclors. The comparability of these PCB data is uncertain and potential effects on risk predictions are unknown (i.e., HQs could be over- or underestimated). PCB measurement basis (i.e., congener, Aroclor or homologue basis) of any additional tissue analyzed will be discussed in the Phase 2 work plan.

PAH exposure for juvenile chinook salmon in the LDW was based on concentrations in juvenile chinook salmon stomach content reported in two studies (McCain et al. 1990; Varanasi et al. 1993) (Section A.4.1.2.2). Because different PAH mixtures were reported in these studies, only the 16 PAHs included in Washington SMS were summed and used to estimate total PAH exposure for this assessment. LDW juvenile chinook salmon stomach content data reported in Varanasi et al. (1993) also included data for a number of alkylated PAHs that were not used to calculate total PAH exposure to juvenile chinook salmon in this risk assessment. Including alkylated PAHs in the total PAH calculation would increase the maximum total PAH concentration reported in Varanasi et al. (1993) from 47 mg/kg ww to 369 mg/kg ww. Inclusion of all PAHs reported (six additional) would increase the maximum total PAH concentration reported in McCain et al. (1990) from 22 to 24 mg/kg ww. Inclusion of all reported PAHs in the total PAH HQ calculation would increase the predicted risk for juvenile chinook salmon. For example, the NOEC-based HQ for growth calculated based on the total PAH concentration (including alkylated PAHs) from Varanasi et al. (1993) data would be 18.[108] However, individual PAHs are not equally toxic to fish for toxic mechanisms other than narcosis, and the TRV is based on BaP alone, so the comparison is highly uncertain. Models to normalize PAH toxicity have been developed for aquatic species (DiToro et al. 2000), and have been applied to fish for use in human health risk assessment (Easton et al. 2002). Use of such models will be further explored in the Phase 2 ERA. Uncertainty regarding comparison of total (of the 16 SMS) PAHs to a BaP-based TRV is discussed further in Section A.7.2.2.3. Because the juvenile chinook salmon PAH HQ was greater than 1, this analysis does not change the risk conclusion for PAHs.

In summary, limited tissue data are a source of uncertainty in the risk conclusions. Based on this uncertainty, the feasibility and utility of collecting additional fish and fish prey tissue data will be discussed in the data gaps memorandum.

Dietary Composition

Dietary composition uncertainty includes lack of data on dietary composition for the ROCs, assumptions used in modeling dietary exposure, and dietary exposure from

[108] Based on a dry weight total PAH concentration of 1,845 mg/kg calculated assuming 20% solids.

sources outside the LDW. Each of these uncertainties is discussed in more detail below.

Very little site-specific data were available to describe the dietary composition of English sole and bull trout feeding in the LDW. It was assumed juvenile chinook salmon consume only amphipods, although it is known they also consume a variety of other benthic invertebrates, zooplankton, larval fish, clam siphons and drift organisms (such as wasps and ants) (Cordell et al. 1996, 1997, 1999). Dietary composition assumptions were important when estimating exposure concentrations of metals and PAHs. The relative importance of this uncertainty is dependent on the relative difference in COPC concentrations in the various prey items that are actually consumed by these fish. Prey that are more closely associated with sediments (e.g., gammarid amphipods) were likely to have higher concentrations of sediment-associated non-biomagnifying chemicals. Additionally, higher-trophic-level prey are likely to have higher concentrations of biomagnifying chemicals, such as mercury and DDTs.

It was assumed that English sole ingest only amphipods, crab, and sediment from the LDW, although they may also consume other benthic invertebrates and larval fish. The dietary risk associated with exposure to arsenic, copper and PAHs may be over- or under-estimated depending on actual chemical concentrations in the English sole diet relative to the amphipod and crab data used to model exposure.

It was assumed that bull trout ingest fish only from the LDW, although they may also consume other prey. In another estuary, bull trout diet was found to contain up to 28% zooplankton (Tokranov and Maksimenkov 1995). Consumption of zooplankton, however, is not likely to increase overall risk from sediment-associated COPCs because the concentrations in algae, the food of zooplankton, is likely lower than that in fish. Zooplankton are low on the food chain, and thus likely to have low body burdens of biomagnifying chemicals relative to fish consumed as prey. Therefore, this uncertainty is not likely to result in higher HQs and does not affect risk conclusions.

Adult shiner surfperch and English sole tissue data were used to model exposure of bull trout to arsenic, copper, chromium, lead, silver, zinc, TBT, mercury, and PCBs.[109] Based on presence of bull trout in the LDW during times of high juvenile fish abundance, bull trout are suspected to primarily consume juvenile fish (Warner and Fritz 1995; Shannon personal communication 2001). Additionally, other piscivorous fish represented by this ROC are not likely to prey on large fish (Miller et al 1977b). It is likely that older fish with longer exposure duration have higher tissue residues of biomagnifying chemicals than juvenile fish. Thus, risk may have been overestimated for these COPCs.

There were no species-specific incidental sediment ingestion data for fish ROCs. As a sensitivity analysis, potential effects of sediment ingestion were estimated by

[109]Note that chromium, lead, silver, and zinc were screened out in the problem formulation.

calculating exposure assuming sediment constituted either 0% or 20% of diet for the English sole (Table A-7-19) to bracket the 10% estimate used in Section A.4.1.2.1.

Table A-7-19. English sole dietary exposure estimates as a function of three sediment ingestion scenarios

COPC	0% Sediment In Diet (mg/kg dw)	10% Sediment In Diet (mg/kg dw)	20% Sediment In Diet (mg/kg dw)
Arsenic	34	32	30
Copper	130	121	114
PAHs[a]	1.7	3.1	3.3

[a] Calculated from wet weight values assuming 20% solids.

If no sediment ingestion is assumed, slightly higher exposure estimates for arsenic and copper were calculated. In contrast, an assumption of a 20% sediment in diet resulted in slightly higher exposure estimates for PAHs. However, HQs did not change substantially for any endpoint (Table A-7-20). Therefore, this uncertainty does not affect the risk conclusions for arsenic, copper, or PAHs.

Table A-7-20. English sole dietary HQs as a function of sediment ingestion

COPC	Endpoint	Worst-case HQ[A]		10% Sed in diet HQ	
		NOEC	LOEC	NOEC	LOEC
Arsenic	survival	na	na	na	na
	growth	**1.7**	**1.1**	**1.6**	**1.1**
Copper	survival	0.18	na	0.16	na
	growth	**16**	**8.0**	**15**	**7.6**
PAHs	survival	0.0007	na	0.0006	na
	growth	0.007	0.03	0.07	0.03

Note: HQs greater than 1.0 are noted in **bold type**.

na – No TRV available

[a] Worst-case HQ is based on the highest exposure estimate for each COPC in Table A-7-18.

In estimating English sole exposure to arsenic and copper, a diet composition of 45% amphipod and 45% crab was assumed. The potential uncertainty associated with this assumption was estimated by conducting a sensitivity analysis, assuming either 100% amphipod or 100% crab consumption. For arsenic, an assumption of 100% crab consumption resulted in slightly elevated HQs for growth (Table A-7-21). For copper, an assumption of 100% amphipod consumption resulted in elevation of the LOEC-based HQ for growth from 7.6 to 10 (Table A-7-21). Therefore, this uncertainty does not affect arsenic or copper risk conclusions for English sole.

Table A-7-21. HQs for English sole as a function of diet

	Dietary HQ							
	Arsenic (assuming 100% crab)		Arsenic (default diet)[A]		Copper (assuming 100% amphipod)		Copper (default diet)	
Endpoint	NOEC	LOEC	NOEC	LOEC	NOEC	LOEC	NOEC	LOEC
Survival	na	na	na	na	0.23	na	0.16	na
Growth	**3.0**	**2.0**	**1.6**	**1.1**	**21**	**10**	**15**	**7.4**

Note: HQs greater than 1.0 are noted in **bold type**.

na – No TRV available

[a] Default diet is 10% sediment, 45% amphipod, and 45% crab.

Site Use

Site use uncertainty includes limited site use data for the ROCs and assumptions regarding site use. These uncertainties are discussed below.

Limited site use data

Limited information on site usage exists for juvenile chinook salmon, bull trout and English sole. It was assumed that LDW fish ROCs use the site 100% of the time, although they may actually forage outside the LDW. This assumption potentially over- or under-estimates risk, depending on the relative magnitude and extent of contamination in other foraging areas. Alternatively, fish may preferentially forage within a select area of the LDW.[110] The dietary exposure analysis would underestimate risk if the actual foraging area were highly contaminated and would overestimate risk if the preferred area were less contaminated. The magnitude of this uncertainty is unknown, but a worst-case scenario (e.g., English sole use a site with 99th percentile concentrations, 100% of the time) was evaluated for English sole below.

Site use assumptions

In Section A.4.1.2.1, SWA surface sediment concentrations were used to model English sole exposure to arsenic and copper. English sole are suspected to forage over a relatively small area of unknown size, although they are also known to migrate seasonally. To address uncertainty associated with foraging range on English sole dietary exposure to arsenic, copper and PAHs, amphipod and sediment components of English sole diet were estimated using the 99th percentile (not spatially weighted) arsenic, copper, and total PAH surface sediment concentrations, rather than the SWA concentrations (Table A-7-22).

[110] There is some evidence that juvenile chinook may preferentially use intertidal habitat.

Table A-7-22. HQs for English sole as a function of estimated surface sediment concentration

Endpoint	Arsenic HQs		Copper HQs		PAH HQs	
	NOEC	LOEC	NOEC	LOEC	NOEC	LOEC
Survival (SWA)	na	na	0.17	na	0.0006	na
Survival (99th percentile)	na	na	0.03	na	0.0007	na
Growth (SWA)	**1.6**	**1.1**	**15**	**7.4**	0.07	0.03
Growth (99th percentile)	**1.8**	**1.2**	**19**	**9.3**	0.15	0.06

Note: Dietary doses assuming 99th percentile sediment concentration of arsenic, copper, and total PAHs are 36, 192, and 36 mg/kg dw, respectively.

HQs greater than 1.0 are noted in **bold type**.

na – No TRV available

Assuming English sole only forage in an area containing the 99th percentile concentration (i.e., only consume sediment and amphipods in the most contaminated area), HQs were slightly higher for arsenic, copper, and PAHs but not substantially different (Table A-7-22). The NOEC- and LOEC-based HQs for copper and growth increased from 15 to 19 and from 7.4 to 9.3, respectively. This analysis suggests that risk estimates calculated assuming that English sole forage over the entire LDW do not significantly underestimate risk. Therefore, uncertainty regarding English sole home range likely does not affect risk conclusions for arsenic, copper, or PAHs.

Potential implications of future restoration projects

A number of habitat restoration projects are underway or planned in the LDW. Successful completion of these projects will result in increased area of mudflats and sandflats, increased riparian structure and productivity, and increased length and complexity of the LDW shoreline (Cordell et al. 2001). Juvenile chinook salmon habitat improvement is the primary goal for most of these projects. However, other fish should also benefit from restoration. Successful restoration in the LDW has been shown to (Cordell 2001a):

- increase diversity and abundance of benthic invertebrates
- increase diversity and abundance of terrestrial insects
- increase extent of low gradient beach and marsh habitat
- may increase diversity and abundance of fish in the LDW

As a result of these potential ecological improvements, location of fish foraging preference may change. These changes may increase or decrease overall risk to fish ROCs, depending on the concentration of sediment-associated chemicals present at restored sites.

Juvenile chinook salmon in the LDW have been shown to have a more varied diet[111] than juvenile chinook salmon in other Pacific Northwest estuaries where chironomid flies (larvae, pupae, and emergent adults) and aphids tend to be the dominant food source (Cordell et al. 2001). Restored marsh vegetation in the LDW has been suggested to increase the plant associated and terrestrial insect prey consumed by juvenile chinook salmon (Cordell et al. 2001). Consumption of more plant and terrestrially derived insects may lead to decreased consumption of aquatic-based benthic prey. Such a shift in diet composition should result in lower overall exposure to sediment-associated COPCs and reduce risk.

A.7.2.2.3 Effects assessment

Uncertainty associated with the available effects data identified in the scientific literature is described in the following sections. Uncertainty associated with test species, laboratory vs. site-specific testing, effects data availability, and safety factors are discussed.

Test Species

There is uncertainty associated with extrapolation of effects from one species to another. It is generally unknown whether species tested in toxicity studies are more or less sensitive to COPCs than LDW receptor species. However, uncertainty should decrease the closer species are taxonomically related. To minimize the potential to underestimate risks, the lowest TRVs for the endpoint of interest for any species were generally selected in this assessment.

Laboratory Testing

The laboratory studies on which TRVs are based were conducted in controlled settings using single-contaminant exposures. Effects associated with multiple-contaminant exposure and other environmental stressors present at the site (e.g., habitat loss) were not factored into these studies. It is unknown if these factors would result in additive, synergistic, antagonistic, or neutral effects on overall risk conclusions.

One method to assess potential effects on fish from exposure to multiple chemicals was presented by Bills et al. (1977, 1981). The first study (Bills et al. 1977) was intended to investigate the potential influence of "background" PCB residues in rainbow trout (*Oncoryhnchus mykiss*) on the sensitivity of the fish to six other chemicals (nitrate, nitrite, cyanide, chlorine, mercury, and chromium) that the fish might encounter in the environment. Experimental fish were first exposed to either 0.01 or 0.1 μg/L (ppb) Aroclor 1254 in a flow-through water system for 30 days, after which subsamples were collected and analyzed for PCB tissue residues. The fish from each exposure regime were then used in a series of acute, static toxicity tests with each of the six other chemicals. LC_{50} values (concentrations producing 50 percent mortality) were determined for each chemical and statistically compared with those from a control

[111] In the LDW, the diet is believed to include different benthic prey, such as clam siphons and marine worms, as well as different terrestrial prey such as wasps and ants.

group of fish that had not been exposed to PCBs. Bills et al. (1977) concluded that the toxicity of four of the six tested chemicals (nitrate, nitrite, chlorine, and mercury) was unaffected by pre-exposure to PCBs. The toxicity of chromium was increased (i.e., it had a significantly lower LC_{50} value) only in the high dose (0.1 µg/L) PCB-exposed fish. The toxicity of cyanide was increased in fish exposed to both PCB doses. The relevance of these findings to an assessment of risks to fish in the LDW is questionable. The maximum total chromium concentration that has been measured in LDW surface water (0.002 mg/L; see Table 4-7 in the RI) is three or four orders of magnitude less than the LC_{50} values (7 to 11 mg/L) reported by Bills et al. (1977). Cyanide concentrations have not been measured in LDW surface water, so it is not known if they would be as high as the LC_{50} values (66 to 90 µg/L) reported by Bills et al. (1977). The mean cyanide concentration in most surface waters is less than 3.5 µg/L (Fiksel et al. 1981, as cited in ATSDR 1997). In water, cyanide occurs most commonly as hydrogen cyanide. Hydrogen cyanide and soluble metal cyanides are removed from water primarily by volatilization (ATSDR 1997).

The second study (Bills et al. 1981) used an identical experimental design to investigate the influence of "background" PCB residues in rainbow trout on their sensitivity to nine chemicals routinely or occasionally used in fishery management. Those chemicals were rotenone, a piscicide; Glidden Durkee 174 (GD-174), a candidate piscicide for carp control; TFM, a lampricide; formalin, a therapeutant; malachite green, a therapeutant; nifurpirinol (Furanace), a therapeutant; Finquel (MS-222), an anesthetic; copper sulfate, an herbicide; and 2,4-D-DMA, an herbicide. Bills et al. (1981) concluded that the toxicity of five of the nine tested chemicals (TFM, formalin, Furanace, MS-222, and copper sulfate) was unaffected by pre-exposure to PCBs. Two of the chemicals (rotenone and 2,4-D-DMA) were more toxic (i.e., had significantly lower LC_{50} values) to the PCB-exposed fish (both doses) than to the control fish. One of the chemicals (GD-174) was more toxic only to the fish pre-exposed to the lower dose of PCBs. One chemical (malachite green) was less toxic (i.e., had significantly higher LC_{50} values) to the PCB-exposed fish than to the control fish. The relevance of these findings to an assessment of effects in the LDW is also questionable. Fishery management chemicals tested by Bills et al. (1981) would be unlikely to be present in the LDW, with the possible exception of 2,4-D-DMA. Although 2,4-D-DMA concentrations have not been measured in LDW surface water, it is unlikely that they are as high as the LC_{50} values (870 to 1,170 µg/L) reported by Bills et al. (1981). The concentration of 2,4-D-DMA rarely exceeds several µg/L in surface water in the absence of a known source such as a spill or application of the herbicide in quantities far in excess of rates typically applied in agriculture or forestry practice (www.speclab.com). Unpublished data from the Green River and its tributaries (upstream of the LDW) show that 2,4-D was detected at concentrations ranging from 0.09 to 0.38 µg/L in 6 of 55 samples collected by King County in 2002 during both baseflow and stormflow conditions. In addition, 2,4-D was detected in only 1 of 24 samples (at a concentration of 0.1 µg/L) collected from the Duwamish River at

Tukwila in 1996 and 1997 (unpublished data from USGS National Water Qualilty Assessment surface water studies).

Regardless of the questionable relevance of the specific results of Bills et al. (1977, 1981) to an assessment of risks to fish in the LDW, such studies evaluating potential effects of multiple chemical exposures are not routinely used in ecological risk assessments. This is because the combined effects of complex chemical mixtures occurring in the environment have not been sufficiently studied.

Basis of PAH TRVs

All available PAH TRVs were based on studies conducted with BaP rather than total PAHs. BaP is frequently used in toxicological studies as a model PAH and is generally believed to be among the most potent carcinogen of the PAH compounds in mammals. However, its relative potency is not known for the specific endpoints evaluated in this ERA.[112] Therefore, the magnitude of this uncertainty is unknown. In addition, there is uncertainty associated with the fact that very few appropriate studies were available for the derivation of PAH TRVs for growth and survival endpoints. Risk associated with PAH exposure will be further assessed in the Phase 2 ERA for juvenile chinook salmon and English sole, and options such as toxic equivalency factors and additivity models may be further explored to compare exposure data with available effects data.

Basis of PCB TRVs for juvenile chinook salmon

The juvenile chinook salmon PCB TRVs were determined following the rationale presented in Section A.4.2. The only endpoints considered were survival and growth; reproductive endpoints are not relevant for juveniles. The selected TRVs for survival and growth were different than the tissue residue effects thresholds presented in a recent National Marine Fisheries Service (NMFS) white paper (Meador et al. 2002) because the white paper had a different goal and the methods employed were different. Meador et al. (2002) conducted a review of research involving PCB exposures and a wide range of endpoints from 15 separate studies. Based on interpretation of these studies, Meador et al. (2002) proposed a lipid-normalized PCB tissue residue effects threshold of 2.4 µg/g lipid. Many of the studies cited by Meador et al. (2002) included sublethal endpoints such as changes in enzyme activity, thyroid hormones, and sensitivity to other chemicals. Effects on growth were noted in only three of the fifteen studies, all at PCB tissue residues much higher than the threshold proposed by Meador et al. (2002). Furthermore, PCB tissue residues were only measured in 8 of the 15 studies; for the others, Meador et al. (2002) estimated tissue residues based on assumed proportions of the exposure concentrations. Only 2 of the 15 studies included mortality as the endpoint, and for those two, Meador et al. (2002) decreased the tissue residue effects thresholds by a factor of 10 to approximate the

[112] With the exception of liver lesions, which were assessed under the mortality endpoint for English sole

threshold for a sublethal endpoint. Although Meador et al. (2002) reported the proposed tissue residue effects threshold on a lipid-normalized basis, only 1 of the 15 studies actually measured the fishes' lipid content; for the other 14 studies, Meador et al. (2002) used an assumed lipid content. All 15 studies cited by Meador et al. (2002) were reviewed for this Phase 1 ERA, and those meeting the criteria outlined in Section A.4.2 were used in the development of TRVs for juvenile chinook salmon (see Table A-4-15). Because the methods used to select TRVs differed between this ERA and the NMFS white paper (Meador et al. 2002), the effects thresholds selected for protection of juvenile chinook salmon also differed. However, the criteria used for selection of TRVs in this ERA are consistent with EPA Superfund guidance, and thus the selected TRVs for survival and growth of juvenile chinook salmon presented in Section A.4.2.6 are considered appropriate for this ERA.

Effects Data Availability

For a number of ROC/COPC pairs, few toxicity data were available to evaluate potential effects on survival, growth and reproduction. This section provides an assessment of associated uncertainty.

Survival TRVs

The following uncertainties in survival TRVs were identified:

- no LOEC or NOEC TRVs were identified for arsenic
- no LOEC TRVs were identified for copper, PAHs, nor mercury

No survival TRV for arsenic was identified for any fish ROC. Fish avoided food at high dietary arsenic concentrations, thus no survival LOEC could be established. However, studies addressing growth endpoints associated with arsenic exposure were believed to be appropriate surrogates for the survival endpoint because fish used in growth experiments presumably had high survival rates throughout the course of the experiment. Thus, growth TRVs for arsenic should be lower, and more conservative, than survival TRVs.

No LOEC TRVs for survival were identified for copper, PAHs, or mercury. All copper, PAH, and mercury NOEC-based HQs were less than 1. Therefore, LOEC-based HQs for survival for these COPCs would also be less than 1.

Growth TRVs

The following uncertainties in growth TRVs were identified:

- weight basis of dietary exposure concentrations were not specified for copper and PAHs studies
- selected TRVs were based on an embryo TRV with a conversion factor to estimate an adult TRV
- only one study was identified that investigated the effects of TBT on growth, and that study had a sediment-based TRV

In the available studies for copper growth (for all ROCs) and PAH growth (for juvenile chinook salmon), dietary exposure concentrations were assumed to represent a dry weight concentration because fish food generally has a low moisture content. However, the weight basis of the dietary concentration in these studies was not reported but was probably wet weight. Effects of this uncertainty were estimated by converting the growth TRVs to dry weight by assuming a 10% food moisture content based on data from Powell et al. (in press) (Table A-7-23). This estimate resulted in an approximately 10% decrease in growth HQs for juvenile chinook salmon for both COPCs, and English sole for copper, suggesting risk may be slightly overestimated for these ROC/COPC pairs. Calculation of HQs using the estimated moisture content did not change the risk conclusions for bull trout.

Table A-7-23. Copper and PAH TRVs and HQs for growth assuming 10% moisture content in food

	NOEC				LOEC			
	TRV mg/kg dw)	HQ (original)	HQ (10% moisture)	HQ % CHANGE	TRV (mg/kg dw)	HQ (original)	HQ (10% moisture)	HQ % CHANGE
Copper								
Juvenile chinook salmon	760	0.24	0.22	-9.2%	778	0.24	0.21	-13%
Bull trout	760	0.01	0.01	0	778	0.01	0.01	0
English sole	8.88	**15**	**13**	-9.5%	17.8	**7.4**	**6.6**	-11%
PAH								
Juvenile chinook salmon	111	**1.7**	**1.5**	-10%	111	0.17	0.15	-10%

Note: HQs greater than 1.0 are noted in **bold type**.

Regarding the use of conversion factors, the PCB growth LOEC for bull trout and English sole (3.72 µg/g ww) was based on a tissue residue reported in eggs. This LOEC was converted to adult tissue residue using an egg to adult conversion factor of 0.43 based on Niimi (1983).There is uncertainty associated with the use of this conversion due to variability in egg to adult ratios compounded by variability among populations and in response to environmental conditions. Thus, based on the uncertainty associated with use of this conversion factor, risk may be over- or underestimated. Because PCB growth HQs were greater than 1 for both English sole and bull trout, this uncertainty does not affect risk conclusions.

Regarding TBT, Hartl et al. (2000) presented the results of a study where 0.4- to 1.8-g wild caught European flounder (*Platichythys flesus*) were exposed to 121 µg/kg TBT (dw) in sediment for 35 days. Length increase of TBT exposed fish was significantly less than that of controls. The results of this experiment are not directly comparable with studies presented in Section A.4.2 because a tissue residue was not presented. However, because sediment concentrations of TBT in the LDW are in the range of

these experimental concentrations, TBT is recommended for further evaluation as a COPC for all fish ROCs.

Reproduction TRVs

The following uncertainties in reproduction TRVs were identified:

- no TRVs were available for arsenic, copper, and PAHs
- few TBT studies were available, and the NOEC was estimated from the LOEC/10
- DDT NOEC was estimated from the LOEC/10
- mercury TRVs were based on an alevin TRV with conversion factors to estimate an adult TRV; NOEC also based on LOEC/10
- mercury reproduction TRVs were higher than the survival TRV

No reproduction TRVs for any fish ROC were identified for arsenic, copper, or PAHs. Because potential reproductive effects may occur at concentrations lower than those where survival or growth effects were observed, risk to fish reproduction in the LDW associated with exposure to these COPCs is uncertain.

For bull trout and English sole, only two studies were available addressing potential reproductive effects associated with a whole-body TBT tissue residue (Table A-4-13). Test species in these studies were Japanese medaka and guppy. No reproduction-based NOEC was available; therefore, no lower bound of toxicity could be determined. The NOEC TRV (0.18 µg/g ww) was estimated by dividing the lowest LOEC (1.79 µg/g ww) by 10, consistent with EPA guidance (EPA 1997).

No DDT reproduction NOEC lower than the selected LOEC was available. Therefore, the reproduction NOEC (0.30 µg/g ww) selected for DDT was based on a LOEC (3.0 µg/g ww) divided by 10. However, the calculated NOEC (0.3 µg/g ww) was lower than the total DDT tissue concentration reported for controls in the experiment from which the LOEC was selected (Macek 1968). Additionally, the magnitude of the effect (approximately 7% mortality in eggs to swim up fry[113]) associated with the selected LOEC was small, but statistically different from controls (approximately 2.5% mortality in eggs to swim up fry). The above assessment suggests that there is uncertainty associated with use of this NOEC; the DDT reproduction NOEC is likely to be greater than the 0.3 µg/g estimated from the LOEC. Thus, risk to the bull trout and English sole ROCs may be overestimated.

To further evaluate the appropriateness of the estimated DDT reproduction NOEC, four additional studies were identified. Because these studies were of field-collected fish with uncontrolled exposures to multiple chemicals, they were not included in the TRV derivation. However, they support the conclusion that a NOEC of 0.3 mg/kg for DDT is likely overly conservative. In these studies, mortalities in fish eggs collected from several different hatcheries and from the field were compared and survival was

[113] Endpoint is an indication of reproductive success.

related to DDT tissue residues accumulated from uncontrolled field or hatchery exposure (Burdick et al. 1964; Cuerrier et al. 1967; Johnson and Pecor 1969; Hopkins et al. 1969). In these studies, LOECs for hatchability ranged from 0.46 µg/g ww for 70% egg mortality in brook trout sac fry (Cuerrier et al. 1967) to 2.9 µg/g ww for lake trout fry (Burdick et al. 1964). Cuerrier et al. (1967) observed no adverse effects in rainbow trout eggs with tissue residues up to 0.18 µg/g ww. Results from Niimi (1983) suggest that a rainbow trout DDT egg tissue residue of 0.18 µg/g corresponds to a maternal (i.e., adult) tissue residue of approximately 0.52 µg/g ww. This analysis suggests the selected reproduction NOEC (0.3 mg/kg) is protective, and the HQ may slightly overestimate risk. Because DDT HQs for English sole were less than 1 (maximum NOEC-based HQ is 0.1) using the available data, this COPC is assumed to pose low risk to English sole. For bull trout, because the maximum HQ is close to 1 (NOEC-based HQ of 0.47), and due to uncertainty in the available tissue residue data (see Section A.7.2.2.3), the feasibility and utility of collecting additional piscivorous fish data will be further addressed in the data gaps memorandum.

Conversion factors were used to determine TRVs for mercury. Thus, the selected bull trout and English sole mercury LOEC and NOEC TRVs (2.1 µg/g and 0.21 µg/g, respectively) for reproduction are highly uncertain. Two conversion factors were used to predict adult tissue residues from alevin tissue data. First, a factor of 3 was applied to convert the rainbow trout alevin mercury concentration to an egg concentration based on a recommendation in Niimi (1983). Second, the egg:adult ratio of 0.05 for rainbow trout reported in Niimi (1983) was applied. There is considerable uncertainty in both of these conversions. Niimi (1983) recommends adjustment to the egg:adult ratio for embryos, but does not comment on alevins. Salmonid alevin live off the yolk sac and are not feeding, so growth dilution may not affect the tissue residue. Additionally, Niimi (1983) notes that the relationship between adult fish tissue residues and egg mercury residue was less well defined than for other contaminants because the proportion transferred from adults to eggs was very low. Conversion of the next lowest LOEC for reproduction (an embryo tissue residue of 0.34 µg/g) would result in a LOEC approximately an order of magnitude higher than the selected LOEC based on the various conversion factors (Section A.4.2.2, Table A-4-12). Due to use of the alevin data with an egg to adult tissue residue correction factor, uncertainty in this TRV is high and risk is uncertain.

TRVs identified for mercury/reproduction were higher than those for survival. Because sub-lethal effects, such as growth and reproduction, are expected to occur at lower doses than effects on mortality, there is uncertainty associated with the use of these TRVs. However, these sublethal TRVs were selected because differences in sensitivity were likely due to differences in species, age, other developmental stages (such as smoltification, sexual development), and environmental conditions (such as source water, temperature, food quality) among the studies identified. Thus, with a limited number of studies available to assess effects associated with each COPC, survival can be found to be a more sensitive endpoint when different conditions or

species are tested. Uncertainty with regards to the selected TRVs may over- or underestimate risks to fish ROCs from mercury.

Safety Factors

As previously discussed, NOECs were not available for all ROC/COPC pairs. Therefore, they were estimated from available LOECs using a safety factor of 10 consistent with EPA guidance (EPA 1997). Of the 31 toxicological studies used in this fish effects assessment that reported both a LOEC and NOEC for growth, survival or reproduction endpoints (available for 6 of the 7 COPCs), the median difference between the LOEC and NOEC was 2.0. Therefore, estimated NOECs likely overestimate risks. The TBT survival LOEC and NOEC were based on safety factors of 5 and 50 based on EPA guidance (EPA 1997) to estimate an LR50. The uncertainty associated with this NOEC estimation is unknown.

A.7.2.2.4 Summary of uncertainties for fish

Uncertainties associated with the fish assessment are summarized in Table A-7-24. The uncertainties with the highest potential to impact risk conclusions were associated with insufficient tissue residue data for English sole and piscivorous fish. Collection of additional fish tissue data is considered highly feasible to fill this data gap.

Table A-7-24. Summary of key uncertainties in fish risk characterization

Issue	Level of Uncertainty	Effect of Uncertainty on Risk Estimate	Potential Means to Decrease Uncertainty	Potential Impact on Risk Conclusions	Feasibility
Exposure Assessment					
Limited English sole tissue data	medium	unknown[a]	collect whole body English sole	high–PCBs; medium-Hg, TBT, DDT	high
No TBT tissue data for juvenile chinook salmon	medium	low to moderate overestimate of risk to juvenile chinook salmon	collect juvenile chinook TBT tissue data	medium- TBT	high
No tissue data for piscivorous fish	high	unknown[a]	collect piscivorous fish tissue data	high–Hg, TBT, DDTs, PCBs	high
Limited bull trout prey data	medium	unknown[a]	collect prey fish tissue	medium- As; low- Cu	high
Limited benthic invertebrate tissue data	high	unknown[a]	collect benthic invertebrate tissue data or stomach contents	high–English sole As; medium–juvenile chinook salmon As, Cu; low–English sole Cu and PAHs and juvenile chinook salmon PAHs	high
Limited dietary composition data	low	unknown[a]	analyze stomach contents	low - juvenile chinook salmon As, Cu; low - English sole As, Cu, PAH	medium
Limited site use data	medium	unknown, depends if preferential feeding	English sole, piscivorous fish tagging studies	medium–bull trout (piscivores); medium-English sole; low-juvenile chinook salmon	low
Effects assessment					
Application of existing TRVs	see Table A-7-25	dependent on applicability of study	additional toxicity testing would be required	see Table A-7-25	low
TRV based on safety factor of 10	medium	potential overestimation of risk	additional toxicity testing would be required	high-bull trout & English sole-TBT high-bull trout-Hg	low

[a] Risk may be higher or lower depending on the concentration of the COPC in the LDW fish population relative to that indicated by the available tissue data.

Level of uncertainty key: **low** = large or relevant dataset
medium = small dataset or limited information
high = very limited data

Potential impact key: **low** = unlikely to result in a change of HQ from less than 1 to greater than 1 (or vice versa)
medium = could result in a change of HQ from less than 1 to greater than 1 if worst-case scenario is used (scenario is viewed as unlikely)
high = HQ could change from less than 1 to greater than 1 (or vice versa) using a scenario that is conservative but more reasonable than the worst-case scenario

Feasibility key: **low** = high budget or difficult research study would be required to address uncertainty
medium = issue could be resolved with a mid-level field sampling event or research study or a detailed assessment of literature
high = issue could be resolved with additional literature search or through limited field sampling

An additional uncertainty was limited data regarding site use by English sole and piscivorous fish species. The feasibility of conducting such studies is relatively low due to the resource-intensive effort that would be required and the difficulty in interpreting such data. The importance of these data is not necessarily to reduce uncertainty in the risk estimates,[114] but rather to provide information to estimate a link between concentrations in fish tissue and concentrations in sediment if needed in the Phase 2 ERA to support management decisions for the site.

There was also uncertainty associated with the risk conclusions for chemicals evaluated using a dietary exposure and effects approach (i.e., arsenic and copper for all three fish ROCs, and PAHs for juvenile chinook salmon and English sole). Limited benthic invertebrate prey tissue data contributed to this uncertainty. This uncertainty could result in either over- or underestimation of risks. Collection of these tissue data is considered feasible to fill this data gap, although because it may be difficult to collect sufficient tissue for analysis in key areas, the feasibility could be somewhat compromised. Additional uncertainty associated with risk predictions for arsenic, copper, and PAHs for all three ROCs is the limited LDW dietary composition data for these fish. However, prey assumptions assessed in the uncertainty assessment did not have a significant impact on risk conclusions.

Reducing uncertainties involving available effects data (Table A-7-25) has a low feasibility because they would generally require additional toxicity testing to verify or supplement toxicological data available in the literature. This type of testing is considered outside the scope of this Superfund ERA.

[114] Because whole body data are used in the critical residue approach, as long as the fish primarily uses the LDW as habitat, these data should integrate exposure over preferred habitat.

Table A-7-25. Summary of uncertainties in TRVs used in fish risk characterization

TRV	Level of Uncertainty	Potential Impact on Risk Conclusions
Arsenic		
Survival	high	low
Growth	medium	low
Reproduction	high	high
Copper		
Survival	medium	low
Growth	medium	low
Reproduction	high	high
Mercury		
Survival	low	low
Growth	low	low
Reproduction	high	high
TBT		
Survival	medium	low
Growth	high	high
Reproduction	high	high
DDTs		
Survival	medium	low
Growth	low	low
Reproduction	high	low
PCBs		
Survival	low	low
Growth	medium	medium
Reproduction	low	medium
PAHs		
Survival	high	medium
Growth	high	high
Reproduction	high	high

Level of uncertainty key: **low** = large or relevant dataset
medium = small dataset or limited information
high = very limited data

Potential impact key: **low** = unlikely to result in a change of HQ from less than 1 to greater than 1 (or vice versa)
medium = could result in a change of HQ from less than 1 to greater than 1 if worst-case scenario is used (scenario is viewed as unlikely)
high = HQ could change from less than 1 to greater than 1 (or vice versa) using a scenario that is conservative but more reasonable than the worst-case scenario

A.7.2.3 Risk conclusions

In the risk estimation, three LOEC-based HQs exceeded 1 for fish ROCs and the growth endpoint (i.e., English sole/copper [7.6]; bull trout/PCBs [2.1]; English

sole/arsenic [1.1]). No other LOEC-based HQs exceeded 1 for any endpoint. Thus, based on available data, the risk was predicted to be greatest for English sole from copper and arsenic and for bull trout from PCBs. NOEC-based HQs were greater than 1 for 12 ROC/COPC pairs. However, as discussed earlier, due to the uncertainty regarding the concentration associated with effects between the NOEC and LOEC, the interpretation of risk based on NOEC-based HQs is more uncertain. The highest NOEC-based HQs were reported for English sole/copper (15) and bull trout/PCBs (8.2). NOEC-based HQs were also greater than 1 for at least one fish species for mercury, PAHs, and TBT.

Due to the small tissue dataset available for the Phase 1 ERA, ROC/COPC pairs to be evaluated in Phase 2 will be determined based on the results of the Phase 1 ERA, the collection and interpretation of data collected to fill data gaps, and the results of the Phase 2 problem formulation. Despite these limitations, Phase 1 risk estimates viewed in the context of the uncertainty discussion provide valuable information for consideration in the data gaps process. In the remainder of this section, risk conclusions for each fish ROC are discussed.

A.7.2.3.1 Juvenile chinook salmon

Juvenile chinook salmon were evaluated as an ROC to represent migratory juvenile salmonids in the LDW, and because as a species, they have been listed as threatened under the Endangered Species Act (Section A.2.3.2.1). Juvenile chinook salmon were evaluated for survival and growth endpoints; reproductive endpoints were not evaluated because juvenile chinook salmon use the LDW only as a migration and rearing corridor, and adult salmon are not believed to have significant exposure to LDW sediments (Section A.2.4.6). Results of the risk characterization for juvenile chinook salmon are summarized in Table A-7-26.

Table A-7-26. Summary of risk characterization for juvenile chinook salmon

COPC	Highest NOEC HQ	Highest LOEC HQ	No. TRV Studies[a]	Uncertainty in TRV[b]	Exposure Data
Arsenic	0.41[c]	0.27[c]	5	medium	modeled amphipod data
Copper	0.24[c]	0.24[c]	9	medium	modeled amphipod data
TBT	**1.1**[d]	0.11[d]	7	medium	wb shiner perch (n=3) (10 fish/composite)
Mercury	0.01[c]	0.009[c]	4	low	wb shiner perch (n=3) (10 fish/composite)
DDTs	0.02[d]	0.01[d]	5	medium	wb juvenile chinook (n=26)[f] (2-10 fish/composite) wb juvenile chinook (n=20) (individual fish)
PCBs	0.004[c]	0.002[c]	5	low	wb juvenile chinook (n=26)[f] (2-10 fish/composite) wb juvenile chinook (n=20) (individual fish)
PAHs	**1.7**[d]	0.17[d]	2	high[e]	juvenile chinook stomach contents (n=8)

Note: HQs greater than 1.0 are noted in **bold type**.

wb - whole body

[a] for all endpoints

[b] uncertainty in TRV for endpoint with highest NOEC HQ. level of uncertainty key: low=large or relevant dataset, medium=small dataset or limited information, high=very limited data

[c] growth endpoint, based on analysis of individual fish

[d] survival endpoint

[e] only two studies available for survival endpoint and fish were dosed with benzo(a)pyrene rather than a mixture of PAHs

[f] three of the composites were statistically constructed from individual samples (by site), as discussed in Section A.4.1.1.

Of the seven COPCs evaluated,[115] only two NOEC-based HQs were greater than 1 for juvenile chinook salmon (Table A-7-26). The NOEC-based HQ for dietary PAH exposure was 1.7 for the growth endpoint and the NOEC-based HQ for TBT was 1.1 for the survival endpoint. No LOEC-based HQs were greater than 1.

Risk to juvenile chinook salmon from PAHs is most uncertain because of the unknown applicability of available TRVs (BaP-based) to the mixture of PAHs that fish are exposed to in the LDW (as represented by the stomach content data). Without resolution of this key uncertainty, additional field data will continue to be difficult to interpret and risks from PAHs will remain uncertain. Additional evaluation of this pair is recommended for the Phase 2 ERA.

Risk to juvenile chinook salmon from TBT was also relatively low using an HQ approach (NOEC-based HQ or 1.1). Both effects and exposure data uncertainties are considered to be medium. However, based on the results of Hartl et al. (2000), potential growth effects cannot be ruled out based on concentrations of TBT in LDW

[115] COPCs were identified for juvenile chinook salmon in Section A.2.4.6 of the problem formulation.

sediments. Based on these results, a different approach for evaluating risk to fish from TBT should be evaluated in Phase 2 (i.e., a sediment-based approach). Therefore, collecting additional TBT tissue data may not be warranted.

Although the HQs calculated for arsenic and copper were less than 1 (NOEC-based HQs were 0.41 and 0.024, respectively), uncertainty due to the paucity of available prey data suggests that additional site-specific prey or stomach content data may be warranted. Only three amphipod composite samples were available and they were collected over a relatively small area. Therefore, collection of additional prey tissue data for juvenile chinook salmon is recommended for Phase 2 to reduce these uncertainties. In addition, for arsenic, regional background concentrations will be addressed in Phase 2, per EPA (2002) guidance.

HQs for mercury, DDT, and PCBs were all at least an order of magnitude less than 1 for juvenile chinook salmon (Table A-7-26). Although uncertainty exists in the exposure and effects assessments for these COPCs (Section A.7.2.2), analysis of the available data suggests that additional field data are unlikely to change the risk conclusions for these pairs.[116]

However, due to continuing agency concerns regarding PCBs, a key site COPC, and juvenile chinook salmon, an ESA species, these data may be considered as a special case, and further analysis is recommended in Phase 2. The HQ conclusions based on literature-based effects data should also be viewed in light of the site-specific work that has been conducted using field-collected juvenile chinook salmon from the LDW. As discussed in Section A.4.3, several studies were conducted in which LDW-collected juvenile chinook were held in the laboratory prior to evaluating their subsequent survival, growth, and ability to withstand immunological challenge (Arkoosh et al. 1998b; Varanasi et al. 1993; Casillas et al. 1995a,b). While these studies have demonstrated elevated exposure to COPCs in the LDW, specifically PCBs, DDTs, and PAHs (Tables A-4-18a, b), the effects observed with these studies have been less conclusive due primarily to fish husbandry and other data interpretation issues, as discussed in Section A.4.3. Therefore, the site-specific studies do not affect risk conclusions for juvenile chinook salmon.

A.7.2.3.2 Bull trout

The bull trout ROC was evaluated to represent piscivores in the LDW for biomagnifying compounds, such as mercury, DDTs, and PCBs, and as a listed species for non-biomagnifying compounds, such as arsenic, copper, and TBT (Section A.4.2). This distinction was made because piscivores may have higher body burdens of biomagnifying chemicals than English sole due to their higher trophic level. To provide a conservative estimate of risk for biomagnifying compounds, in the calculation of HQs, toxicological data available for the most sensitive fish species

[116] To reach NOEC-based HQs of 1, mercury and DDT concentrations in prey would have to be 98 and 44 times greater, respectively.

tested for these COPCs were compared to exposure concentrations estimated in upper-trophic-level fish species (using a predator-prey factor). Results of the risk characterization for bull trout are summarized in Table A-7-27. No site-specific studies have been conducted with bull trout or any other piscivore, so risk characterization is based on literature effects data only.

Table A-7-27. Summary of risk characterization for bull trout

COPC	Highest NOEC HQ	Highest LOEC HQ	No. TRV Studies[a]	Uncertainty in TRV[b]	Exposure Data
Arsenic	0.29[c]	0.19[c]	5	medium	wb shiner perch (n=3, 10 fish/composite)
Copper	0.01[c,d]	0.01[c]	9	medium	wb shiner perch (n=3, 10 fish/composite)
TBT	**1.1**[d]	0.11[d]	7	high[f]	wb shiner perch (n=3, 10 fish/composite)
Mercury	**2.2**[d]	0.94[d]	15	low	modeled from wb shiner perch[f] (n=3, 10 fish/composite)
DDTs	0.37[g]	0.04[g]	12	high[h]	modeled from wb juvenile chinook salmon[i] (n=26, 2-10 fish/composite)[k]
PCBs	**8.2**[c]	**2.1**[c]	16	medium	modeled from wb English sole[j] (n=3, 20 fish/composite)

Note: HQs greater than 1.0 are noted in **bold type**.

wb - whole body

[a] for all endpoints

[b] uncertainty in TRV for endpoint with highest NOEC HQ. level of uncertainty key: low=large or relevant dataset, medium=small dataset or limited information, high=very limited data

[c] growth endpoint

[d] survival endpoint

[e] only two studies available for reproduction endpoint

[f] maximum measured perch tissue multiplied by a PPF of 5

[g] reproduction endpoint

[h] only two studies available for reproduction endpoint. Available data suggest TRV is conservative (see section A.4.2.5.2 and A.7.2.2)

[i] 95% UCL concentration in juvenile chinook salmon multiplied by a PPF of 3.5

[j] maximum English sole tissue concentration multiplied by a PPF of 3.5

[k] three of the composites were statistically constructed, as discussed in Section A.4.1.1.

HQ Results for Biomagnifying COPCs

For biomagnifying COPCs, the LOEC-based HQs exceeded 1 only for PCBs (2.1 for growth) (Table A-7-27), indicating the potential for adverse effects from PCBs. NOEC-based HQs exceeded 1 for mercury (maximum LOEC-based HQ of 2.2) and PCBs (maximum LOEC-based HQ of 8.2) but was less than 1 for DDT (Table A-7-27). Because HQs were greater than 1 for PCBs and mercury, these COPCs are recommended for further evaluation in the Phase 2 ERA, and collection of additional piscivorous fish tissue analysis is recommended for Phase 2.

Because the exposure data are highly uncertain (Table A-7-27), additional fish tissue analysis of DDT is also recommended, despite the uncertainties in the effects data.

HQ Results for Non-biomagnifying COPCs

- No LOEC-based HQs exceeded 1 for non-biomagnifying COPCs (arsenic, copper, TBT; Table A-7-27).[117] TBT had a NOEC-based HQ equal to 1 (maximum NOEC-based HQ of 0.99; Table A-7-27). The highest NOEC-based HQs for arsenic and copper were 0.29 and 0.013 for growth, respectively (Table A-7-27).
- Available exposure and effects data for TBT were limited, and thus the risk prediction is uncertain. Collecting additional fish tissue (as prey) for TBT analysis would partially reduce this uncertainty. However, the sediment-based approach suggested in Hartl et al. (2000) with flounder (see Section A.7.2.2.3) should also be considered in Phase 2.
- Although the HQs calculated for arsenic were less than 1 (maximum NOEC-based HQ of 0.29), the risk from arsenic is also uncertain. Uncertainty in the exposure analysis due to the limited available prey data suggests that additional site-specific data may be warranted. Therefore, collection of additional prey tissue data for piscivorous fish is recommended for Phase 2. In addition, for arsenic, regional background concentrations will be addressed in Phase 2, per EPA (2002) guidance.
- HQs for copper were two orders of magnitude less than 1. Although uncertainty exists in the exposure and effects assessments for copper (see Section A.7.2.2), collection of additional copper data in prey fish tissue is unlikely to change the risk conclusions. The effects assessment was based on several studies that addressed copper toxicity to fish of the family Salmonidae, and the exposure assessment was conservatively based on copper concentrations in adult fish as prey. Therefore, copper appears to pose low risk to bull trout in the LDW, based on available data.

A.7.2.3.3 English sole

The English sole ROC was evaluated to represent all fish in the LDW not specifically covered by juvenile chinook salmon or bull trout. English sole are highly exposed to sediment-associated contaminants based on their close proximity and diet of benthic invertebrates. To provide a conservative estimate of risk from COPCs in the calculation of HQs, toxicological data from the most sensitive fish species tested for these COPCs were compared to exposure concentrations measured or estimated in English sole. Results of the risk characterization for English sole are summarized in Table A-7-28.

[117] PAHs, another non-biomagnifying COPC for fish, were not evaluated for bull trout because PAHs do not accumulate in fish tissue, and thus have an incomplete pathway to bull trout (see Section A.7.2.2).

Table A-7-28. Summary of risk characterization for English sole

COPC	Highest NOEC HQ	Highest LOEC HQ	No. TRV Studies[a]	Uncertainty in TRV[b]	Exposure Data
Arsenic	**1.6**[c]	1.1[c]	6	medium	wb English sole (n=3, 20 fish/composite)
Copper	**15**[c]	**7.6**[c]	11	medium	wb English sole (n=3, 20 fish/composite)
TBT	0.11[d,e]	0.01[d,e]	7	high[f]	wb English sole (n=3, 20 fish/composite)
Mercury	0.38[e]	0.16[e]	15	low	wb English sole (n=3, 20 fish/composite)
DDTs	0.02[d]	0.002[d]	12	high[g]	English sole fillet (n=9, 5-20 fish/composite)
PCBs	**2.4**[c]	0.62[c]	16	medium	wb English sole (n=3, 20 fish/composite)
PAHs	0.07[c]	0.03[c]	3	high[h]	wb English sole (n=3, 20 fish/composite)

Note: HQs greater than 1.0 are noted in **bold type**.

wb - whole body

[a] for all endpoints

[b] uncertainty in TRV for endpoint with highest NOEC HQ. level of uncertainty key: low=large or relevant dataset, medium=small dataset or limited information, high=very limited data

[c] growth endpoint

[d] reproduction endpoint

[e] survival endpoint

[f] only two studies available for reproduction endpoint

[g] only two studies available for reproduction endpoint. Available data suggest TRV is conservative (see Section A.4.2.5.2)

[h] only three studies were available for the growth endpoint and fish were dosed with benzo(a)pyrene rather than a mixture of PAHs

Two LOEC-based HQs exceeded 1 for English sole. For the growth endpoint, LOEC-based HQs of 1.1 and 7.6 were calculated for arsenic and copper, respectively (Table A-7-28). NOEC-based HQs for growth exceeded 1 for arsenic and copper (1.6 and 15, respectively) and also for PCBs (maximum NOEC-based HQ of 2.4). All other HQs were less than 1, as discussed below.

Because exposure to arsenic and copper was estimated to be greater than that associated with effects, additional assessment of these pairs is recommended in Phase 2. These pairs were evaluated using a dietary approach, therefore, collection of additional prey tissue data for English sole is recommended for Phase 2. Note also that regional background issues with arsenic will be addressed in Phase 2, per EPA (2002) guidance.

Based on available data, there is risk of adverse effects from PCBs. To reduce uncertainty in the whole-body concentration of PCBs in English sole, additional tissue analysis is recommended, and further evaluation is proposed for the Phase 2 ERA.

Although HQs were less than 1 for mercury (maximum NOEC-based HQ of 0.38), based on uncertainties in the whole-body English sole tissue data, additional tissue analysis for mercury is recommended, and further evaluation is proposed for the Phase 2 ERA.

The maximum NOEC-based HQ for TBT was 0.21 (Table A-7-28), based on whole-body concentrations in English sole and relatively uncertain tissue effects data. The sediment-based TRV, suggested in Hartl et al. (2000) for flounder, may provide less uncertainty than collection of additional tissue data. This approach should be further explored in Phase 2.

The highest NOEC-based HQ for DDT was 0.023 (Table A-7-28). This HQ was based on fillet data collected from throughout the LDW, and on conservatively estimated TRVs. Because concentrations of DDT in English sole fillets are unlikely to be an order of magnitude less than whole-body concentrations (Section A.7.2.2.3), risks from DDT to English sole appear to be low based on available data. Therefore, collection of additional tissue data to evaluate this pair is unlikely to change the risk conclusion.

Site-specific studies were available to provide additional perspective regarding risk to English sole in the LDW.[118] Increased exposure to COPCs in the LDW, such as PCBs and PAHs, has been documented (Casillas et al. 1991; Stein et al. 1992; Collier et al. 1992; Johnson et al. 1988; Myers et al. 1998; Sections A.4.1.2.2, A.4.3.2). Malins et al. (1984) and Rhodes et al. (1987) reported increased lesion prevalence in LDW-collected English sole relative to reference sites. However, Johnson and Landahl (1994) suggest that increased lesion prevalence has not affected mortality rates or age structure in fish from contaminated sites including the LDW. Results from several studies suggest that some reproductive measures in English sole collected in the LDW may be impaired relative to fish from reference sites (Johnson et al. 1988, 1993, 1997; Casillas et al. 1991; Section A.4.3.2.3). Linking the results of field studies to risks from specific chemicals is difficult considering, among other factors, the complex mixtures of chemicals in the field and the uncertainties in English sole home range. In addition, interpreting cause and effect of the adverse effects observed in field studies is complicated due to a variety of potential variables including genetic variation, health, or seasonal variation in the spawning cycle. The only COPC with an HQ greater than 1 for reproductive effects in English sole was PCBs, with a NOEC-based HQ of 1.2. No TRVs with a reproductive endpoint were available for PAHs. However, using statistical techniques based on correlations, Johnson et al. (1988, 2002) have suggested that PAHs may be partially responsible for reduced reproduction observed in English sole from contaminated sites. Therefore, risks from PCBs and PAHs to English sole will be further evaluated in the Phase 2 ERA, and the techniques used in these studies will be reviewed in detail.

A.7.2.3.4 Fish risk conclusion summary

In summary, based on a synthesis of the HQ calculations presented in the risk estimation and the uncertainty assessment for fish, the following Phase 1 recommendations were made, based on available data. Note that the list of analytes to be measured in Phase 2 data collection will be identified in the Phase 2 work plan, and

[118] These site-specific studies are relevant to English sole itself, not necessarily to English sole as a surrogate for other fish.

the final COPC list for the Phase 2 ERA will be determined in the Phase 2 Problem Formulation.

- **Juvenile chinook salmon.** TBT and PAHs are recommended for further evaluation in the Phase 2 ERA. Gathering additional arsenic and copper exposure data is recommended in Phase 2. Mercury, DDT, and PCBs were estimated to pose low risk. However, PCBs are recommended for further evaluation in the Phase 2 ERA due to the ESA status of juvenile chinook salmon and identification of PCBs as a key site COPC for other ROCs.
- **Bull trout.** TBT, mercury and PCBs are recommended for further evaluation in the Phase 2 ERA. Gathering additional arsenic and DDT exposure data is recommended in Phase 2. Copper was estimated to pose low risk, based on available data.
- **English sole.** Arsenic, copper, PCBs, TBT, and PAHs are recommended for further evaluation in the Phase 2 ERA. Additional mercury exposure data is recommended in Phase 2. DDT was estimated to pose low risk, based on available data.

A.7.3 Risk Characterization for Wildlife

This section presents an estimation of risk by calculating HQs for each of the five wildlife ROCs using dietary exposure doses (and egg concentrations for heron) along with effects TRVs presented in Section A.5.2. Following the risk estimation, a detailed evaluation of uncertainty associated with these calculations is presented. Finally, this section presents a risk conclusion that integrates HQ results with associated uncertainty to summarize the results of the Phase 1 risk assessment based on available data, and provides input to the data gaps process. ROC/COPC pairs to be evaluated in the Phase 2 ERA will be determined in the Phase 2 problem formulation following a process described in the Phase 2 work plan; pairs selected for further evaluation in Phase 2 will be based on the results of the Phase 1 ERA and on interpretation of data collected during Phase 2.

A.7.3.1 Risk estimation

This section presents the HQ calculations for wildlife ROC/COPC pairs. Dietary dose HQs are presented in Table A-7-29 and egg concentration HQs for heron are presented in Table A-7-30. Results for each ROC are described in the sections below.

Table A-7-29. Dietary dose HQs for wildlife ROC/COPC pairs

COPC	Exposure Dose (mg/kg bw/day)	NOAEL TRV (mg/kg bw/day)	LOAEL TRV (mg/kg bw/day)	NOAEL HQ	LOAEL HQ
Sandpiper					
PCBs	0.363	0.41	0.94	0.88	0.39
Copper	27.5	47	62	0.59	0.44
Lead	8.23	2.0	20	**4.1**	0.41
Zinc	23.9	82	123	0.29	0.19
BEHP	0.419	5.1	350	0.08	0.001
Great blue heron					
Lead	0.0825	2.0	20	0.04	0.004
Mercury	0.0156	0.0091	0.091	**1.7**	0.17
PCBs	0.109	0.41	0.94	0.27	0.12
Bald eagle					
Lead (SUF=0.25)	0.0104	2.0	20	0.005	0.0005
Lead	0.0415	2.0	20	0.02	0.002
Mercury (SUF=0.25)	0.0026	0.0091	0.091	0.28	0.03
Mercury	0.0103	0.0091	0.091	**1.1**	0.11
PCBs (SUF=0.25)	0.0298	0.41	0.94	0.07	0.03
PCBs	0.119	0.41	0.94	0.29	0.13
River otter					
PCBs	0.128	0.015	0.15	**8.5**	0.85
Arsenic	0.774	0.126	1.26	**6.1**	0.61
Lead	0.0619	0.5	1.5	0.12	0.04
Harbor seal					
PCBs (SUF=0.33)	0.0103	0.015	0.15	0.69	0.07

Note: HQs greater than 1.0 are noted in **bold type**.

SUF – site use factor. SUFs range from 0 (species does not use the site) to 1 (species uses this site exclusively); all SUFs were assumed to equal 1 unless otherwise indicated.

Table A-7-30. Egg HQs for heron

COPC	Egg Concentration (mg/kg ww)	NOEC TRV (mg/kg ww)	LOEC TRV (mg/kg ww)	NOEC HQ	LOEC HQ
PCBs	47	7.1	16	**6.6**	**2.9**
TEQs	1.8×10^{-3}	0.5×10^{-3}	1×10^{-3}	**3.6**	**1.8**

Note: HQs greater than 1.0 are noted in **bold type**.

TEQ – Summation of toxicity equivalence factors (TEFs) multiplied by the corresponding concentration of PCB congeners

A.7.3.1.1 Spotted sandpiper

Five COPCs were evaluated for sandpiper: PCBs, copper, lead, zinc, and BEHP. No HQs associated with observed effects doses were greater than 1, and only one NOAEL-based HQ exceeded 1. Lead had a NOAEL-based HQ of 4.1 (Table A-7-29).

A.7.3.1.2 Great blue heron

COPCs evaluated for great blue heron were lead, mercury, and PCBs. Risks to heron from PCBs were evaluated by two methods due to the availability of heron egg data collected from the colony in West Seattle. This section discusses both lines of evidence for PCBs (dietary dose approach and concentrations in eggs). PCBs in eggs were evaluated as total PCBs and as 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) equivalents (TEQs), which were calculated from the PCB congener data. TEQs are the sum of the dioxin-like toxicity of the PCB congeners, and are expressed as a single concentration equivalent to the toxicity of a similar concentration of TCDD. The other two COPCs for heron were evaluated from a dietary dose perspective only.

Using the dietary dose approach, no LOAEL-based HQ exceeded 1. Mercury was the only COPC with a NOAEL-based HQ exceeding 1 for great blue heron (1.7) (Table A-7-29). Using the egg data, in which only PCBs were measured, the LOEC-based HQ for PCBs (2.9), and TEQs (1.8) exceeded 1 (Table A-7-30), and the egg NOEC-based HQs (6.6 for PCBs and 3.6 for TEQs) were greater than 1. Note that HQs for PCBs were less than 1 for great blue heron using the dietary approach. Uncertainties in both approaches are discussed in Section A.7.3.2.

A.7.3.1.3 Bald eagle

HQs for bald eagle were calculated for lead, mercury, and PCBs using two alternative exposure doses: one dose was calculated using a highly conservative SUF of 1, and the other dose was calculated using a lower SUF of 0.25. This range of SUFs was used because of limited information regarding site use of the LDW by bald eagles. No LOAEL-based HQs exceeded 1 for bald eagle. The only eagle HQs exceeding 1 based on either SUF (Table A-7-29) was mercury, with a NOAEL-based HQ of 1.1, using a SUF of 1. The NOAEL-based HQ for mercury was proportionally lower (0.29) with the SUF of 0.25.

A.7.3.1.4 River otter

Three COPCs were evaluated for river otter; PCBs, arsenic, and lead. No LOAEL-based HQs exceeded 1 for river otter. However, the NOAEL-based HQs for both PCBs and arsenic exceeded 1 (8.5 and 6.1, respectively) (Table A-7-29).

A.7.3.1.5 Harbor seal

PCBs were the only COPC evaluated for harbor seal in the wildlife exposure and effects assessment (Section A.5), based on the screens presented in Section A.2.4.7.2. The NOAEL- and LOAEL-based HQs were both less than 1 (0.69 and 0.07, respectively).

A.7.3.2 Uncertainty assessment

This section presents a discussion of uncertainty associated with the problem formulation and the exposure and effects assessments for wildlife ROCs. An uncertainty is considered to have the potential to significantly impact risk conclusions

if alternative calculations or relatively small changes in exposure data could result in a change of an HQ from less than 1 to greater than 1, or vice versa. At the end of this section, key areas of uncertainty are summarized and qualitatively ranked as low, medium, or high according to their level of uncertainty, potential to impact risk conclusions, and overall feasibility of reducing uncertainty either through literature-based analysis or field studies.

A.7.3.2.1 Problem formulation

Primary uncertainties in the problem formulation were associated with ROC selection, exposure pathways, and the COPC screen, as discussed below in the following sections.

ROC Selection

While numerous bird and mammal species use the LDW, five organisms (i.e., spotted sandpiper, great blue heron, bald eagle, river otter, and harbor seal) were selected as ROCs to evaluate risk from sediment-associated chemicals in the LDW. The wildlife ROC selection process was presented in Section A.2.3.3. Uncertainty associated with selected wildlife ROCs and their ability to serve as protective surrogates for other species are discussed below.

Spotted sandpiper

Spotted sandpipers were selected to represent benthivorous birds expected to be highly exposed to COPCs based on their consumption of benthic invertebrate species and relatively significant rate of incidental ingestion of sediment. Other sediment-probing birds at the site include western sandpiper, dunlin, and dowitcher. The sediment ingestion rate used for spotted sandpiper is expected to be a reasonable representation of other LDW sediment-probing birds (Norman 2002). Dabbling ducks such as mallard or American widgeon may also ingest sediment, but published sediment ingestion rates for mallard were not as high as those for sandpiper species (EPA 1993b). Section A.7.3.2.2 of this uncertainty section includes an evaluation of the sensitivity of the risk calculations to sediment ingestion rates.

Estimated sandpiper exposures should be greater than those of herbivorous birds, such as American coot, American widgeon, mallard, and geese that use the LDW because COPC exposure through ingestion of invertebrates for birds represented by sandpiper is likely to be higher than exposure through ingestion of plants. Exposure is a function of COPC concentrations in prey, and the availability of the prey item for consumption. Plants are not widely available in the LDW. Although future habitat restoration may increase the availability of plants, and thus increase the presence of herbivores, sandpiper are likely to be more highly exposed because of assumed higher COPC concentrations in invertebrates than in plants (especially plants in restored areas). Therefore, estimated sandpiper exposure should be greater than exposure of herbivorous birds. In addition, killdeer, a primarily insectivorous bird, is also likely to

be less exposed to LDW sediment-associated COPCs than sandpiper because killdeer obtain most of their food from terrestrial sources.

Spotted sandpipers may differ from other benthivorous birds in their daily food consumption (DFC) rates relative to their body weight. A greater body-weight-normalized DFC would result in higher dietary exposure of COPCs, thus leading to greater risk. Based on information presented in EPA (1993b), sandpipers have a higher DFC rate than other waterfowl, such as lesser scaup, mallard, and Canada goose, so risk to sandpiper would overestimate risk to those species. Sandpipers were assumed to ingest only amphipods, although sandpiper or other benthivorous birds may also consume some fish, crab, or mussels. Section A.7.3.2.2 of the uncertainty section addresses an alternative dietary composition for sandpipers.

Great blue heron and eagle

Great blue herons and eagles were selected to represent piscivorous and carnivorous birds in the LDW, respectively. Piscivorous birds in the LDW include loons, western grebe, mergansers, double-crested cormorant, pigeon guillemot, Caspian tern, common murre, and osprey. An uncertainty in using heron and eagle to represent piscivorous birds is their consumption of different prey types. This uncertainty is addressed by calculating exposure using maximum COPC concentrations in any fish species (Section A.7.3.2.2, subsection Dietary Composition), as opposed to the HQ estimate presented in Table A-7-29, which used perch data for heron and a combination of fish for eagle. Other omnivorous species such as bufflehead, Barrow's goldeneye, and surf scoter may consume primarily mussels, clams, or crab. To evaluate the relative exposure of piscivorous and omnivorous birds, exposure of heron was calculated assuming a diet of mussels and crabs in Section A.7.3.3.2. Peregrine falcons are carnivores that primarily consume other birds. Dietary exposure of eagles may not represent that of peregrine falcons if the quantity of birds consumed from the LDW differs. No data were available to quantify this difference, but it was assumed that eagles are more exposed because peregrine falcons are likely to feed extensively on pigeons in urban areas. Therefore, from a dietary preference perspective, eagles are likely the most exposed carnivorous bird found in the LDW.

Great blue herons and bald eagles may have different normalized DFC rates than other species they are representing, resulting in different exposure to COPCs. DFC rates for other piscivorous waterfowl, seabirds, and raptors in the LDW were reviewed in EPA (1993b) and Nagy et al. (1999). Information for species found in the LDW was identified only for osprey, although data were found for four other species similar to those in the LDW (herring gull, black guillemot, common tern, and thick-billed murre). All five species had higher normalized DFCs than great blue herons and eagles (46 and 26 g dw/kg bw/day, respectively); common tern had the highest rate (159 g dw/kg bw/day) and osprey had the lowest rate (47 g dw/kg bw/day). The results of substituting these other species' DFCs for the eagle 's and heron's are shown in Table A-7-31. For three species (thick-billed murre, black guillemot, and common

tern), risk conclusions for mercury and PCBs would change based on an increase in the HQ from less than one to greater than or equal to one. Although these results indicate that some species in the LDW (common murre, pigeon guillemot, and Caspian tern) could be more highly exposed than great blue heron or eagle, the likelihood of this exposure is low based on infrequent site usage. In extensive bird surveys conducted by Cordell et al. (1996, 1997, 1999, 2001) and Canning et al. (1979), pigeon guillemot and common murre were rarely observed.[119] Caspian tern was not observed during the year-long survey by Canning et al. (1979) and was observed in two of four surveys by Cordell et al. (1999 and 2001), although frequency of observation during those two surveys was not available. None of these species nest near the LDW, so they would not be exposed during sensitive life stages. In summary, although some species of piscivorous seabirds in the LDW have high food consumption rates, chemical exposures of heron and eagle are expected to be higher than those seabirds because of their more frequent use of the site. Therefore, these ROCs adequately represent other piscivorous birds.

Table A-7-31. NOAEL HQs calculated using the daily food consumption rate for common tern as compared to those calculated in the original risk estimation in Section A.7.3.1.

ROC/COPC	NOAEL HQ using DFC for other species[a]					
	Original NOAEL HQ	Osprey	Herring Gull	Thick-billed Murre	Black Guillemot	Common Tern
Heron						
Lead	**0.04**	0.05	0.06	0.10	0.13	0.16
Mercury	**1.7**	1.9	**2.5**	**4.2**	**5.4**	**6.5**
PCBs	0.27	0.29	0.38	0.66	0.85	**1.0**
Eagle						
Lead (SUF=0.25)	0.005	0.008	0.01	0.02	0.02	0.03
Lead (SUF=1)	0.02	0.03	0.04	0.08	0.10	0.12
Mercury (SUF=0.25)	0.28	0.46	0.60	**1.0**	**1.3**	**1.6**
Mercury (SUF=1)	**1.1**	**1.8**	**2.4**	**4.1**	**5.3**	**6.3**
PCBs (SUF=0.25)	0.07	0.11	0.15	0.26	0.33	0.39
PCBs (SUF=1)	0.28	0.46	0.60	**1.0**	**1.3**	**1.6**

Note: HQs greater than 1.0 are noted in **bold type**.

[a] DFC was the only parameter that was changed from original HQ calculations (i.e., SUF and dietary assumptions for heron and eagle were used for the other species)

[119] Common murre was observed by Canning et al. (1979) on only two occasions during the year-long survey; only one bird was seen on each occasion. Pigeon guillemot was not seen by Canning et al. (1979), and was observed during only one of four LDW surveys by Cordell et al. (1999).

River otter and harbor seal

Mammals using the LDW are represented by the river otter and harbor seal ROCs, both of which are primarily piscivorous. Risk estimates for river otter, representing semi-aquatic mammals, are likely to be greater than those for muskrat and raccoon because otters are more exposed to sediment-associated chemicals via food consumption from the LDW. Muskrats feed on plants, which are not abundant throughout the LDW, and raccoons feed on a greater proportion of terrestrial prey. Harbor seals represent marine mammals, which include California sea lions and harbor porpoises. All three species are opportunistic feeders, primarily of fish and some invertebrates. The uncertainty associated with the assumption that harbor seal prey composition represents that of sea lion and porpoise is addressed below in Section A.7.3.2.2, which uses the maximum COPC concentration detected in any fish type (which was higher than concentrations measured in invertebrates) to characterize risk. Based on the above, river otters are expected to have greater exposure and harbor seals are expected to have similar or greater exposure to LDW sediment-associated chemicals than other mammals using the LDW.

Pathways

The dermal exposure pathway for birds and mammals was considered to be complete, but there were no toxicological data available to evaluate the significance of this pathway. The extent to which elimination of this pathway may underestimate risk is not known, but is likely to be insignificant relative to risk associated with ingestion of contaminated prey. Four exposure pathways for wildlife were considered complete but of unknown significance (see Figure A-2-3). These include: 1) eagle and bird ingestion, 2) sandpiper and fish ingestion, 3) sandpiper and other ingestion (terrestrial insects and mollusks), and 4) seal and benthic ingestion. The consumption of birds by eagles is addressed in Section A.7.3.22 under tissue data, and the other three exposure pathways are discussed in the same section under dietary consumption.

COPC Screen

The list of COPCs for the King County (KC) wildlife risk assessment (King County 1999c), which was a key component of the COPC screen for wildlife in this Phase 1 ERA, did not include pesticides. An additional conservative screen was conducted for exposure of piscivorous birds and seals to DDT in Section A.2.4.7. Based on the available data, the results of this screen indicated that risks from DDT to these ROCs were low. However, risk to wildlife from other pesticides is unknown.

In the KC wildlife risk assessment, risk to ROCs was based on exposure data from both Elliott Bay and LDW, which may have underestimated risk if LDW-only exposure concentrations were higher than Elliott Bay and LDW concentrations combined, as discussed below. This uncertainty applies to sandpiper, eagle, and otter; for great blue heron the KC assessment calculated HQs for heron fledglings using LDW-only data.

For eagle and otter, fish (i.e., perch and salmon) were assumed to constitute most of the diet (99% and 98%, respectively) and were the risk-driving components in the King County wildlife risk assessment for COPCs other than lead. Arsenic, copper, mercury, zinc, and TBT were screened out in the King County assessment. However, this assessment was based on combined tissue data from Elliott Bay/LDW for perch (salmon data were from LDW only). Therefore, risks for the LDW alone may have been underestimated for those COPCs if LDW perch tissue concentrations of these COPCs were sufficiently higher than those in Elliott Bay. A comparison of these COPCs in LDW-only vs. Elliott Bay/LDW tissue data are shown in Table A-7-32. Given that the 95th percentile HQs for these COPCs from the KC assessment were low (all were less than 0.13, except for copper and otter, which was 0.39), the use of LDW-only tissue data would not shift any of these HQs above 1.

Table A-7-32. Metal concentrations in perch from LDW samples and from combined Elliott Bay/LDW samples

	Perch Tissue Concentrations (mg/kg ww)			
	LDW Only		Elliott Bay/LDW	
COPC	Mean	Standard Error	Mean	Standard Error
Metals/Metalloids				
Arsenic	0.127	0.0117	0.121	0.00594
Copper	1.44	0.378	1.09	0.231
Mercury	0.0779	0.00521	0.0533	0.0113
Zinc	18.1	0.436	17.6	0.309
Organometallics				
Tributyltin	0.153	0.018	0.140	0.0146

For spotted sandpiper, King County used amphipod data from LDW only and sediment data from both LDW and Elliott Bay. The incidental sediment ingestion component of the dose for sandpipers included both LDW and Elliott Bay data. However, risk was driven primarily by ingestion of invertebrates for all COPCs screened out by King County, except arsenic. For arsenic, the SWA sediment concentration in the LDW was 10 mg/kg dw, slightly higher than the Elliott Bay/LDW concentration of 6.3 mg/kg dw used by King County. This difference of less than a factor of 1.6 is not likely to increase the 95th percentile HQ for arsenic (0.29) to greater than 1, although the HQs could not be calculated because the King County probabilistic model was not accessible.

Based on this analysis, the COPC screen appropriately identified the ROC/COPC pairs, with the possible exception of pesticides. It is unknown whether pesticides may be posing risk, although the analysis with available DDT data indicated low risk to great blue heron and bald eagle in the LDW. Exposure data for DDT were not available for spotted sandpiper.

A.7.3.2.2 Exposure assessment

Uncertainty in the exposure assessment was associated with calculating the exposure dose for each ROC including daily food consumption, dietary composition, site use factor, and COPC concentrations in prey and sediment. These uncertainties are discussed in detail below.

Daily Food Consumption

Species-specific DFC rates were not available for spotted sandpiper, great blue heron, or river otter. Consumption rates for these ROCs were calculated one of two ways: 1) using data for a similar species, or 2) using allometric equations developed from data for a related group of birds or mammals. The level of uncertainty in these calculations is likely to increase as the group of birds or mammals used to develop the allometric equation widens and encompasses a greater taxonomic variety of organisms. For spotted sandpiper, the consumption rate was calculated using data for the common sandpiper. For great blue heron, an allometric equation for wading birds was applied. Thus, for these two birds, uncertainty introduced by the use of these allometric equations should be low. Greater uncertainty applied to the river otter's DFC rate, which was calculated using an allometric equation developed by Nagy (1987) for non-herbivorous placental mammals. However, the river otter DFC would need to increase from 0.26 kg dw/day to 2.1 kg/dw/day for the lead NOAEL HQ to increase from 0.12 to 1. It is unlikely the DFC rate would be more than four times higher than currently calculated, so risk conclusions should not be significantly affected for lead. River otter NOAEL HQs for PCBs and arsenic are already greater than 1, so an increase in the DFC rate would not affect risk conclusions for those COPCs.

Dietary Composition

Uncertainty associated with the site-specific dietary preferences of LDW ROCs may result in lower or higher estimates of exposure. Very few site-specific data were available for ROCs feeding in the LDW. It was assumed that spotted sandpipers eat only amphipods, although they may also consume other benthic invertebrates, fish, mussels or crab. The only prey items that would increase the sandpiper exposure dose for any of the COPCs were crab, which would increase zinc exposure, and fish, which would increase PCB exposure. Using the unlikely assumption that crab constitutes 25% of the sandpiper diet, the LOAEL-based HQ for zinc would increase from 0.19 to 0.32, resulting in no effect on risk conclusions. Similarly, using the unlikely assumption that perch constitutes 25% of the sandpiper diet, the LOAEL-based HQ for PCBs would increase from 0.88 to 1.0. This conservative assumption would change the risk conclusion for sandpiper/PCBs, but it is unlikely.

It was assumed that great blue herons ingest only fish from the LDW, although they may also consume crustaceans or mollusks. In addition, the omnivorous birds they represent, such as surf scoter, may consume crab and mussels. Using a conservative assumption that heron consume 50% perch and 50% crab (or mussels, whichever had the higher concentration), the NOAEL-based HQ for lead would increase from 0.041 to

0.065, the NOAEL-based HQ for PCBs would decrease from 0.27 to 0.23, and the NOAEL-based HQ for mercury would increase from 1.7 to 2.1. These slight differences would not have changed risk conclusions.

The proportions of different types of fish consumed by heron, eagle, otter, and seal are not known. Risk could be underestimated if higher proportions of more contaminated fish were consumed by ROCs. To evaluate this uncertainty, the maximum concentration of a COPC measured in any fish (perch, sole, or salmon) was used in calculating exposure. The resulting NOAEL- and LOAEL-based HQs are shown in Table A-7-33. The only NOAEL-based HQ that increased from less than 1 to greater than 1 (when based on elevated PCB concentrations in English sole) was for seal/PCBs, which increased from 0.69 to 1.5. This analysis could affect risk conclusions, and is discussed in Section A.7.3.3.

Table A-7-33. HQs calculated assuming fish component of diet contained maximum concentration detected in any fish type compared to HQs calculated for original risk estimation in Section A.7.3.1

COPC	NOAEL HQ MAX	NOAEL HQ ORIGINAL	LOAEL HQ MAX	LOAEL HQ ORIGINAL
Great blue heron				
Lead	0.04	0.04	0.004	0.004
Mercury	**1.7**	**1.7**	0.17	0.17
PCBs	0.95	0.27	0.41	0.03
Bald eagle				
Lead (SUF=0.25)	0.005	0.005	0.0005	0.0005
Lead (SUF=1)	0.02	0.02	0.002	0.002
Mercury (SUF=0.25)	0.29	0.28	0.03	0.03
Mercury (SUF=1)	**1.2**	**1.1**	0.12	0.11
PCBs (SUF=0.25)	0.16	0.07	0.07	0.03
PCBs (SUF=1)	0.64	0.29	0.28	0.13
River otter				
PCBs	**18**	**8.5**	**1.8**	0.85
Arsenic	**7.7**	**6.1**	0.77	0.61
Lead	0.13	0.12	0.04	0.04
Harbor seal				
PCBs	**1.5**	0.69	0.15	0.07

Note: HQs greater than 1.0 are noted in **bold type**.

SUF – site use factor. SUFs range from 0 (species does not use the site) to 1 (species uses this site exclusively); all SUFs were assumed to equal 1 unless otherwise indicated.

In addition, species-specific incidental sediment ingestion rates were uncertain for ROCs. For sandpipers, measured ingestion rates for four species other than spotted sandpiper ranged from 7 to 30% of the diet, so an average of these values was used. Table A-7-34 shows the difference in HQs when calculated using the highest

sandpiper ingestion rates of 30% None of the NOAEL- or LOAEL-based HQs that were below 1 would become higher than 1, so risk conclusions would not change. For other ROCs with relatively low sediment ingestion rates, HQs were recalculated assuming a very high sediment ingestion rate of 10%. HQs changed by only slight amounts and risk conclusions would not change, as shown in Table A-7-34. Therefore, the sediment ingestion rates do not result in significant uncertainty for identifying COPCs for the Phase 2 ERA.

All three bird ROCs may obtain a portion of their diet from terrestrial sources. However, this assessment assumed 100% of ROC diets are composed of aquatic prey, which may overestimate exposure to LDW-related COPCs.

Table A-7-34. NOAEL HQs calculated using higher sediment ingestion rates compared to those calculated in the original risk estimation in Section A.3.7.1

	Original NOAEL HQ	NOAEL HQ with Higher Sediment Ingestion Rate
Sandpiper[a]		
PCBs	0.88	0.92
Copper	0.59	0.61
Lead	**4.1**	**4.7**
Zinc	0.29	0.32
BEHP	0.08	0.08
Heron[b]		
Lead	0.04	0.14
Mercury	**1.7**	**1.8**
PCBs	0.27	0.27
Eagle[b]		
Lead (SUF=0.25)	0.005	0.03
Lead (SUF=1)	0.02	0.10
Mercury (SUF=0.25)	0.28	0.29
Mercury (SUF=1)	**1.1**	**1.2**
PCBs (SUF=0.25)	0.07	0.07
PCBs (SUF=1)	0.29	0.30
River otter[b]		
PCBs	**8.5**	**8.6**
Arsenic	**6.1**	**6.4**
Lead	0.12	0.58
Harbor seal[b]		
PCBs (SUF=0.33)	0.69	0.69

Note: HQs greater than 1.0 are noted in **bold type**.

SUF – site use factor. SUFs range from 0 (species does not use the site) to 1 (species uses this site exclusively); all SUFs were assumed to equal 1 unless otherwise indicated.

[a] 30% sediment ingestion rate used for higher exposure scenario

[b] 10% sediment ingestion rate used for higher exposure scenario

Site Usage

The percentage of food captured from LDW by river otters is uncertain, although a study in New York found the home range of river otters ranged from 1.5 to 22 km (Spinola et al. 1999, as cited by EPA 1993b). This distance compares to the length of the LDW of about 8.5 km (5 mi). The exposure assessment assumed a site use factor of 1, a conservative assumption that may overestimate risk if river otter also forage in areas outside of the LDW.

A conservative site use factor of 1 was also applied to herons, although it is not certain if they feed only within LDW based on site-specific observations. Within the LDW, great blue herons have been observed feeding in the vicinity of Kellogg Island, where they are more likely to obtain their food than other areas of the LDW (Krausmann 2002a). The fish data on which the dietary residues were based were collected from this area of the river, reducing the uncertainty associated with use of available fish data with respect to heron site usage. Sediment was assumed to be a small component of the heron's diet, so risk from sediment ingestion is not likely to be a significant factor in overall risk.

The source of PCBs in heron eggs is assumed to be from maternal transfer. The extent of maternal exposure to LDW COPCs is not certain for the heron eggs collected from the West Seattle colony. Based on observations of individual birds from the West Seattle colony it was estimated that at least half of the colony used the LDW to forage, focusing primarily on the Kellogg Island area (Krausmann 2002a). Note, however, that no successful nesting occurred there in 2000 or 2001 (Norman 2002a), and there are indications this colony may have been abandoned. If females from which the eggs were collected foraged in areas other than the LDW, calculated risk based on egg concentrations related to LDW exposure could be underestimated or overestimated, depending on the relative PCB contamination at these sites.

Spotted sandpipers are likely to have a feeding range substantially smaller than the size of the LDW. Although limited information is available to estimate the potential size of the area, it has been estimated at 1.5 km (1 mi) along the LDW (Norman 2002b). Sandpipers ingest a relatively large amount of sediment (compared to the other ROCs), so the area in which sandpipers feed and the concentration of COPCs within that area may have a strong influence on the outcome of the exposure estimate. This uncertainty is influenced by the sediment data used in exposure calculations, as discussed below in the sediment data uncertainty section.

A site use factor of 0.33 was assumed for harbor seal based on a site-specific survey along with conservative assumptions (see Section A.5.1). For eagle, a range of site use factors was applied to exposure calculations because of very limited information about their use of the site for feeding. Use of the most conservative site use factor resulted in an increase in the NOAEL -based HQs from 0.28 to 1.1 for bald eagle/mercury.

Therefore, uncertainty in the SUF may significantly impact the risk estimate for this ROC/COPC pair, as described in Section A.7.3.3.

Tissue Data

A small number of tissue samples were collected, particularly for amphipods, crab, perch, and sole, where numbers of samples ranged from two to four. These samples were composites of 3 individuals for crab, 10 for perch, 20 for English sole, and about 2,000 for amphipods. Although the numbers of samples were low, composite samples incorporate variability in individuals and thus provide a larger picture of average exposure. However, uncertainty would be lower with a greater number of composite samples. This uncertainty may over- or underestimate exposure for all ROCs.

Amphipods were collected near Kellogg Island in areas of low-to-moderate sediment concentrations for most COPCs. Because sandpipers have been observed feeding in other areas of the LDW that have higher sediment concentrations of some COPCs, risk may be underestimated using available amphipod tissue concentrations. Risk from PCBs, for example, could be underestimated because of the higher sediment PCB concentrations in areas such as the eastern shoreline from RM 2.8 and 3.8 relative to those near Kellogg Island (see Map A-7-1). A relatively small increase in amphipod tissue PCB concentration from the measured value of 2.2 to a potential value of 2.5 mg/kg dw would result in a NOAEL-based HQ of 1. Copper in amphipods would need to increase from 166 to 290 mg/kg dw, and zinc from 132 to 500 mg/kg dw, to result in NOAEL-based HQs equal to one. Because of the possibility that PCBs, copper, and zinc concentrations could be high enough in invertebrate prey to result in risk, these uncertainties are discussed further in Section A.7.3.3.1.

Crab, shiner surfperch, and English sole were collected in the lower portion of the LDW in the vicinity of Kellogg Island. There is uncertainty regarding the movement of these species within LDW and whether these tissue residue data represent fish from other areas of the LDW. Shiner surfperch are found throughout the LDW (Miller 1977a), but site-specific home range information was not available. English sole are most abundant in the lower portion of the river (based on otter trawl data), although they are seasonally present throughout the LDW (Miller 1977a). Juvenile Dungeness crab and adult red rock crab are likely to be present near Kellogg Island, where the available data were collected, but could also occur further upstream.[120] The PCB HQs are the most likely to be affected by these uncertainties because areas with higher PCB concentrations were found farther upstream in the LDW (see Map A-7 -1). The extent to which this uncertainty might underestimate risk is not known.

Piscivorous birds are a component of bald eagle's diet, but tissue data were not available. Concentrations of PCBs and mercury in piscivorous birds may be higher than in prey fish tissue because these COPCs biomagnify in higher trophic level

[120] Efforts to collect adult crab upstream of Kellogg Island up to RM 4 were unsuccessful because no adult crab were observed or caught (ESG 1999). Juvenile crab were caught up to RM 2.1.

organisms. Therefore, risk to eagle may be underestimated assuming a diet of only fish. This uncertainty is addressed by recalculating HQs using estimated bird tissue concentrations and assuming birds constitute 19% of the diet (see discussion of eagle diet composition in Section A.5.1.3.3). To address this uncertainty, the PCB concentration in LDW birds was estimated using a mean biomagnification factor (BMF) of 93 reported in the literature for total PCBs from alewife to whole-body herring gulls (Braun and Norstrom 1989). Assuming a fish tissue concentration of 4.13 mg/kg dw (mean of perch, sole and salmon data presented in Table A-5-3 for eagle), the estimated PCB concentration in birds was calculated as 384 mg/kg dw. The PCB NOAEL-based HQs would increase from 0.07 and 0.29 with SUFs equal to 0.25 and 1, respectively, to 1.3 and 5.4. LOAEL-based HQs would be 0.58 and 2.3, respectively. It should be noted that these HQs are very conservative because they assume that birds ingest only fish from the LDW.

To estimate mercury concentrations in birds, a BMF of 9.2 from fish to eagle was used. This BMF was calculated from mercury concentrations in prey fed to osprey nestlings and the nestling's tissue (DesGranges et al. 1998). On average, 90% of the mercury in various tissue types was in the methylmercury form. Assuming a fish concentration of 0.35 mg/kg dw (mean of perch and sole data presented in Table A-5-3), the estimated mercury concentration in birds would be 3.2 mg/kg dw/day. Using this estimated concentration and assuming birds constitute 19% of eagle's diet, the mercury NOAEL-based HQ at a SUF=1 would increase from 1.1 to 2.5, and at a SUF=0.25 would increase from 0.28 to 0.71. Although this uncertainty may have an impact on HQ calculations, it does not change overall risk conclusions.

As previously discussed in the fish uncertainty section (Section A.7.3.2.2), some whole-body English sole samples were missing livers and all were missing portions of other tissue. The estimated maximum whole-body PCB concentration could increase to 2.7 mg/kg ww, or 11 mg/kg dw, based on calculations presented in the uncertainty section for fish (Section A.7.2.2), assuming a moisture content of 76%. Using this concentration in calculations would increase risk only slightly; for example, the NOAEL-based HQ for eagle with SUF=1 would increase from 0.29 to 0.33. Risk conclusions would not be impacted for any of the ROC/COPC pairs.

The age class of salmon for which whole-body data were available (juveniles) may not represent that consumed by eagles, otters, or seals. Older fish are more likely to have higher concentrations of bioaccumulative compounds in their tissues. However, adult salmon would likely constitute only a small portion of the ROCs diet based on their prey size preferences. Adult salmon returning to LDW probably range from 50 to 100 cm in length (estimated from information in Groot and Margolis [1991]), whereas eagles[121] and seals prefer fish less than about 30 cm, and otters prefer fish less than about 40 cm (see discussion of ROC diet composition in Section A.5.1.3). This

[121] Although eagle may also consume larger dead or dying fish, 90% of fish captured by eagles foraging at Hood Canal and Indian Island were less than 30 cm long (Watson and Pierce 1998).

uncertainty may slightly underestimate risk from PCBs and mercury, but risk conclusions would not likely change.

Crab data are uncertain because whole-body samples were not analyzed; instead weighted average concentrations estimated from edible meat and hepatopancreas data were used. River otter was the only ROC assumed to ingest crab. However, because crab was estimated as only 10% of its diet, this uncertainty is not likely to change risk conclusions for otter COPCs.

There is uncertainty in the heron egg data because the result from the sample with the highest concentration was estimated using assumptions about proportional weights of the two subsamples. However, this uncertainty would not change risk conclusions because the HQs would remain above 1 for both total PCBs and PCB-TEQs (4.2 and 1.5) using the lowest concentration detected in either subsample (30 mg/kg for total PCBs and 0.077 mg/kg PCB-TEQs). An additional uncertainty in the heron egg data is associated with the non-standard HPLC/PDA methods used for analysis. In addition, avian TEFs were not available for congeners 170 and 180 to calculate TEQs, so mammalian TEFs were used instead. Depending upon the difference in avian and mammalian TEFs and the comparability of the results generated using the nonstandard analytical method to more standard GC/ECD methods, this could result in an over- or underestimation of risk.

Sediment Exposure Data

For spotted sandpiper, SWA sediment data from all intertidal locations were used in the risk calculations. However, because sandpipers have a small foraging range, it is possible they could feed from a smaller area within the LDW that could be more contaminated than the entire intertidal area. A range of about 1.5 km along the LDW for nesting or migratory sandpipers was considered a reasonable estimate (Norman 2002b). The distributions of all COPCs, except PCBs and BEHP (Maps A-7-1 and A-7-2), were relatively evenly distributed in the intertidal areas such that SWA COPC concentration using 1.5 km of shoreline in any particular area would not be substantially different than using all intertidal areas. To address the uncertainty in PCB and BEHP exposure, SWA sediment concentrations were calculated for the intertidal river area from RM 2.8 to RM 3.8 (including Slip 4), which contains the highest concentrations of these COPCs. These calculations resulted in the following increase in risk: the NOAEL-based HQs increased from 0.88 to 0.97 for PCBs and from 0.082 to 0.083 for BEHP, and did not change any risk conclusions. This uncertainty is discussed further in Section A.7.3.3.1.

Spatially weighted sediment averages used in bird risk calculations were not calculated for all intertidal areas because the low sampling density in some areas would provide higher relative weight to those concentrations that could result in disproportionate weighting of some measurements and potentially bias the average values. To evaluate the uncertainty in excluding these areas, SWAs were recalculated using mean concentrations in the excluded areas and assuming these areas were 20%

of the total intertidal area. These new SWAs were then used in the calculations for sandpiper, the ROC most exposed by the sediment ingestion pathway. HQs for all sandpiper COPCs, except lead, did not increase by more than 0.02 HQ units; the lead HA increased from 4.1 to 4.2. Therefore, this uncertainty would not affect risk conclusions for birds. For ROCs other than sandpiper, SWA sediment concentrations from all intertidal areas or all areas of the LDW were used to represent exposure from incidental sediment ingestion. These values may not represent actual exposure if ROCs forage preferentially in specific areas rather than evenly throughout the LDW or intertidal areas. However, this uncertainty does not significantly affect risk characterization because sediment ingestion is a very small component of the diet for these ROCs.

Restoration Project Implications

A number of habitat restoration projects are underway or planned in the LDW. Improved habitat from restoration may increase wildlife use of the LDW, resulting in higher proportions of food obtained from the LDW and possibly higher exposure to COPCs. However, current risk calculations already include site use factors of 1 for sandpiper, heron, eagle, and otter. It is not known if increased abundance of fish would raise the site use factor of 0.33 for seal, but it appears unlikely because of the relatively small foraging area of the LDW relative to other nearby areas in Elliott Bay and Puget Sound.

Additionally, locations of wildlife foraging preference may change. These changes should decrease risk to wildlife ROCs, assuming the concentrations of sediment-associated chemicals in restored sites are lower than the SWA concentrations used for current risk calculations.

A.7.3.2.3 Effects assessment

Uncertainty associated with the available effects literature may also affect risk characterization, as described below in the following sections.

Test Species

None of the laboratory toxicological studies used for deriving wildlife TRVs were conducted using ROC species, so there is uncertainty in extrapolating effects from one species to another. Uncertainty is probably the least for PCBs and mammals because of the numerous studies conducted with mink and the likelihood that mink are at least as sensitive as river otters or seals (Section A.5.2.3). For effects of arsenic and lead on mammals, and copper on birds, data were only available for common laboratory species and not wildlife species, so uncertainty may be greatest for these ROC/COPC pairs. It is not known if species used in laboratory studies are more or less sensitive to COPCs than LDW receptor species, so risk may be over- or underestimated.

There are a number of studies showing that reproductive endpoints in chickens are particularly sensitive to PCBs when compared to other bird species (Kennedy et al. 1996), as discussed in Section A.5.2.1.1. Because of the likelihood that chicken PCB

TRVs would overestimate risk to wildlife (EPA 2000b), they were not used to calculate HQs. The lowest observed PCB dose with a reproductive effect (decreased egg production and fertility) in chickens was 0.35 mg/kg bw/day (Platonow and Reinhart 1973), compared to the wildlife LOAEL used in this assessment of 0.94 mg/kg bw/day. There was no dose associated with no effect in the study by Platonow and Reinhart (1973). If 0.35 mg/kg bw/day were used as the LOAEL TRV, the sandpiper LOAEL-based HQ would increase from 0.39 to 1.0, but the LOAEL-based HQs for heron and eagle would remain below 1.

Egg production in Japanese quail appears to be a very sensitive endpoint for lead, with a LOAEL of 0.2 mg/kg bw/day. This concentration resulted in a 20% decrease in egg production compared to the control (approximate change from 6 to 5 eggs/hen/week). Because, like chickens, Japanese quail are raised specifically for their egg production capability, it is uncertain that this effect is relevant to reproductive effects in wildlife species. Therefore, the egg hatchability endpoint for Japanese quail, with a LOAEL of 20 mg/kg bw/day was used to estimate risk to LDW ROCs. Use of the sensitive quail endpoint would increase all bird HQs for lead by three orders of magnitude, and would affect risk conclusions.

Laboratory vs. Site-Specific Testing

The laboratory studies used to derive TRVs were conducted in controlled settings using single-contaminant exposures. Effects from multiple-contaminant exposure and other environmental stressors present at the site were not factored into these studies. It is not known if these factors would result in additive, synergistic, antagonistic, or neutral effects on risk.

Effects Data Availability

Laboratory effects data were more limited for some ROC/COPC pairs than others, possibly resulting in greater uncertainty in those TRVs. In particular, data for BEHP and birds were very limited. Only one study, conducted with chickens, resulted in an effect at the lowest dose tested, which was a relatively high concentration compared to the available NOAEL data. However, the LOAEL-based HQ for birds and BEHP using this effect concentration was very low (0.0012), so even a decrease in the LOAEL of one to two orders of magnitude would not significantly increase risk. Other ROC/COPC pairs with relatively limited toxicological data were copper and zinc for birds (based on growth in chicks), and arsenic for mammals. It is not known if these limited data result in over- or under-estimation of risk for those ROC/COPC pairs.

Safety Factors

For some NOAELs, laboratory data were not available, so they were estimated from LOAELs using a factor of 10. For birds, this approach was used for mercury, and for mammals it was used for PCBs, arsenic, and lead. The uncertainty associated with the NOAEL estimation is unknown, although the true NOAEL may be only slightly lower than the experimental LOAEL (Sample et al. 1996). Of the 14 studies evaluated in this

risk assessment with both NOAEL and LOAEL pairs, 11 of the pairs had NOAELs that were 1.3 to 5 times lower than the LOAEL. The remaining three differed by a factor of 10, because the dose levels differed by a factor of 10. It is possible that the difference may have been lower if a smaller interval in doses had been used. These data indicate that the safety factor of 10 is a conservative estimate of the potential difference between the NOAEL and LOAEL, and therefore, may overestimate risk.

A.7.3.2.4 Summary of uncertainties for wildlife

Uncertainties associated with the wildlife assessment are summarized in Table A-7-35. The uncertainties with the highest potential to impact risk conclusions are associated with sandpiper. For this ROC, uncertainties related to site use and amphipod tissue data could result in an over- or underestimation of ingestion of COPCs, and therefore, may affect risk conclusions. Collection of additional amphipod data was considered feasible to reduce this uncertainty, although the feasibility could be somewhat compromised because it may be difficult to collect sufficient tissue for analysis in key areas. A site use assessment would likely require a higher level of effort.

For eagle, prey selection and COPC concentrations in fish tissue are the major uncertainties that may affect risk conclusions. The potential for ingestion of other birds by eagles to impact the risk conclusion is considered medium for PCBs because an HQ greater than 1 would result only by using the worst-case assumption that birds consumed by eagles feed entirely on fish resident to the LDW. The feasibility of collecting birds for tissue chemical analysis was given a medium rank because of the level of effort and possible permitting constraints for some species. Fish tissue data collection was considered highly feasible. The uncertainty related to eagle/mercury is due to the SUF, and was given a medium rank because the HQ would equal 1 only if the highly conservative assumption that eagles feed entirely on LDW prey was used.

For heron and otter, the primary uncertainties are related to limited fish tissue data and unknown proportion of fish types in the diet. Using a worst-case scenario of heron ingesting only English sole containing the maximum detected PCB concentration resulted in an HQ of 1, so this uncertainty was ranked as medium. For otter, the PCB HQ was greater than 1 (8.5) and using worst-case-scenario fish concentrations would increase the HQ to 18, but would not change risk conclusions. The feasibility of filling the fish tissue data gap was considered high, while determining the proportion of fish in the heron's or otter's diet would require a more substantial field effort.

For seal, key uncertainties are associated with diet composition and fish tissue data. For PCBs, a HQ of 1.5 was calculated assuming that seals obtain 33% of their food from the LDW[122] and consume only English sole containing the maximum detected concentration (a conservative assumption). Therefore, the potential to impact risk conclusions was ranked medium. The feasibility of collecting additional fish data was

[122] A SUF for seal of 0.33 is based on the observation of seals 17 times in the 52 days surveyed in the LDW (WDFW 1999)

considered high, but feasibility of gathering information on diet composition was low because of the permitting issues and extended fieldwork that would be necessary.

Table A-7-35. Summary of primary uncertainty in wildlife risk characterization

Issue	Level of Uncertainty	Effect of Uncertainty on Risk Estimate	Potential Means to Decrease Uncertainty	Potential Impact on Risk Conclusions	Feasibility of Filling Data Gap
Exposure Assessment					
Fish consumption by sandpiper	medium	slight underestimate of risk	observe feeding habits of sandpiper in the LDW	low	low
Bird ingestion by eagle	medium	underestimate of risk assuming birds contain higher COPC concentrations than fish	collect bird data; model tissue concentrations in bird	medium for PCBs, low for mercury and lead	medium
Proportion of fish types in piscivore diets	high	underestimate of risk assuming ROCs consume higher proportions of more contaminated fish	observe feeding habits of heron; analysis of stomach contents, scat samples, or observe feeding habits for seal	medium for heron/PCBs and seal/PCBs, low for others	low
Site usage by eagle	medium	potential underestimate of risk if lower site use factor is used	conduct eagle site use survey	medium for mercury, low for PCBs and lead	low
Amphipod tissue data and site usage for sandpiper	medium	potential underestimate of risk	collect additional amphipod tissue data, conduct sandpiper site use survey	high for PCBs, medium for copper and zinc	high, medium
Fish tissue data	high	unknown[a]	collect additional fish tissue data	medium for PCBs/eagle, PCBs/heron and PCBs/seal, low for otter	high
Daily food consumption rate of otter	medium	unknown; risk could be over- or underestimated	measure daily consumption rates of otter	low	low
Effects Assessment					
Application of available effects data	see Table A-7-36	dependent upon applicability of study	additional toxicity testing would be required	see Table A-7-36	low

[a] Risk may be higher or lower depending on the concentration of the COPC in the LDW fish population relative to that indicated by the available tissue data.

Level of uncertainty key: **low** = large or relevant dataset
medium = small dataset or limited information
high = very limited data

Potential impact key: **low** = unlikely to result in a change of HQ from less than 1 to greater than 1 (or vice versa)
medium = could result in a change of HQ from less than 1 to greater than 1 if worst-case scenario is used (scenario is viewed as unlikely)
high = HQ could change from less than 1 to greater than 1 (or vice versa) using a scenario that is conservative but more reasonable than the worst-case scenario

Feasibility key: **low** = high budget or difficult research study would be required to address uncertainty

medium = issue could be resolved with a mid-level field sampling event or research study or a detailed assessment of literature

high = issue could be resolved with additional literature search or through limited field sampling

In addition to factors related to exposure, there is uncertainty regarding laboratory effects data, as summarized and ranked in Table A-7-36. The TRVs with the highest uncertainty levels, and the highest potential impact on risk conclusions are those for PCBs, copper, and zinc in birds and lead in mammals. Uncertainty associated with the BEHP TRV for birds is high, but would not likely affect the very low HQ. Uncertainty with the arsenic TRV for mammals is high, but may not affect risk conclusions based on a NOAEL-based HQ of 6.1. Uncertainty associated with the remaining TRVs is either low or would have a low impact on risk conclusions.

Table A-7-36. Summary of uncertainty in TRVs used in wildlife risk characterization

TRV	Level of Uncertainty	Potential Impact on Risk Conclusions
Birds		
PCBs – eggs	medium	low
PCBs – diet	medium	high
Copper	high	high
Lead	medium	low
Mercury	medium	low
Zinc	high	high
BEHP	high	low
Mammals		
PCBs	low	low
Arsenic	high	medium
Lead	medium	high

Level of uncertainty key: **low** = large or relevant dataset
medium = small dataset or limited information
high = very limited data

Potential impact key: **low** = unlikely to result in a change of HQ from less than 1 to greater than 1 (or vice versa)
medium = could result in a change of HQ from less than 1 to greater than 1 if worst-case scenario is used (scenario is viewed as unlikely)
high = HQ could change from less than 1 to greater than 1 (or vice versa) using a scenario that is conservative but more reasonable than the worst-case scenario

A.7.3.3 Risk conclusion

None of the dietary LOAEL-based HQs exceeded 1 for any wildlife ROC. Thus, based on existing data, risks to wildlife appear to be low. NOAEL-based HQs (Table A-7-29) were greater than 1 for five ROC/COPC pairs. However, as discussed earlier, due to the uncertainty regarding the concentration associated with effects between the NOAEL and LOAEL, the interpretation of risk based on NOAEL-based HQs is more uncertain. The highest NOAEL-based HQs were reported for river otter/PCBs (8.5), river otter/arsenic (6.1), and sandpiper/lead (4.1). NOAEL-based HQs were also just

over 1 for mercury and great blue heron and eagle, assuming a site use factor (SUF) of 1.

However, risks associated with PCBs measured in heron eggs from a nest in West Seattle indicated a higher potential for risk for PCBs than the dietary HQs. The NOEC- and LOEC-based HQs based on this line of evidence were greater than 1 (6.6 and 2.9, respectively) (Table A-7-30). Using a TEQ approach to assess the PCB risk to great blue heron with the egg data also resulted in LOEC-based HQs greater than 1 (3.6 and 1.8, respectively). These results are contradictory to the HQs calculated for great blue heron and PCBs using the dietary approach, where LOAEL- and NOAEL-based HQs were less than 1 (0.12 and 0.27, respectively). However, considering the uncertainty in these estimates using either approach, differences in risk estimates of this magnitude are not large. It appears that exposure of great blue heron to PCBs in the LDW area may be near the level where adverse effects on reproduction could occur.

Due to the small tissue dataset available for the Phase 1 ERA, ROC/COPC pairs to be evaluated in Phase 2 will be determined based on the results of the Phase 1 ERA, the collection and interpretation of additional data in Phase 2, and the results of the Phase 2 problem formulation. Despite these limitations, Phase 1 risk estimates viewed in the context of the uncertainty discussion provide valuable information for consideration in the data gaps process. In the remainder of this section, risk conclusions for each wildlife ROC are discussed.

A.7.3.3.1 Spotted sandpiper

Spotted sandpiper was chosen to represent benthivorous birds such as dunlin, dowitcher, western sandpiper, and dabbling ducks. Thus, risk characterization for sandpiper should be protective of other benthivorous birds because of sandpiper's high exposure to COPCs through ingestion of benthic invertebrates and incidental sediment. Results of the risk characterization for spotted sandpiper are summarized in Table A-7-37.

Table A-7-37. Summary of risk characterization for spotted sandpiper

COPC	NOAEL HQ	LOAEL HQ	No. of TRV Studies	Uncertainty in TRV	Exposure Data [a]
PCBs	0.88	0.39	6	medium	amphipod (n=4, 2000 organisms/ composite)
Copper	0.59	0.44	3	high [b]	amphipod (n=4)
Lead	**4.1**	0.41	5[c]	medium	amphipod (n=4)
Zinc	0.29	0.19	4	high [d]	amphipod (n=4)
BEHP	0.08	0.001	3	high [e]	amphipod (n=4)

Note: HQs greater than 1.0 are noted in **bold type.**

[a] amphipod composite samples were collected only at Kellogg Island

[b] growth endpoint in chicks

[c] not including three studies with chicken or quail egg production endpoint

[d] endpoints were growth or mortality in chickens, or mortality in mallards

[e] only one study noted effects; this study exposed chickens at only high doses

No LOAEL-based HQs exceeded 1 for any of the sandpiper COPCs (i.e., PCBs, copper, lead, zinc, and BEHP). Therefore, based on the available data, exposure to these COPCs in the LDW is not predicted to occur at concentrations associated with adverse effects. However, the NOAEL-based HQ for lead (4.1) was greater than 1, so depending on the threshold for effects between the NOAEL and LOAEL, effects due to lead are possible. The key uncertainty associated with the lead assessment was the limited amphipod (prey) data available to estimate exposure. Therefore, collection of additional amphipod data from intertidal sandpiper habitat is recommended for Phase 2 to reduce uncertainties in the sandpiper/lead assessment, and this pair is recommended for further evaluation in the Phase 2 ERA.

Collection of additional benthic invertebrate prey data (represented by composite sampling of infaunal/epifaunal invertebrates) is also recommended to reduce uncertainties in the sandpiper/PCB assessment.[123] Additional sandpiper site usage information would also be valuable in reducing uncertainties in the sandpiper/PCB pair. Although the NOAEL-based HQ for PCBs was less than 1 (0.88) based on available data, a small increase in PCB concentrations in sandpiper prey would result in an HQ greater than 1. In addition, preferential feeding in areas of the LDW with higher PCB concentrations than those near the West side of Kellogg Island would also result in HQs greater than 1. Therefore, additional data are needed to reduce these uncertainties.

For copper and zinc, NOAEL HQs were less than 1, but there is uncertainty associated with both the TRV and the amphipod tissue data, based on the small number of available samples. If additional amphipod data were collected resulting in an increase in the current 95% UCL copper tissue concentrations from 166 to 290 mg/kg dw and zinc from 132 to 500 mg/kg dw, NOAEL-based HQs would equal 1. Although there were no copper or zinc hotspots in the river that would indicate that amphipod concentrations are likely to be higher in other areas, it is recommended that if additional amphipod data are collected for further characterization of risk to sandpiper, copper and zinc should also be measured.

The NOAEL-based HQ for BEHP was much less than 1 (0.082). Alternative exposure calculations based on uncertainty in the available data are not expected to increase the HQ to 1, and although uncertainty in the TRV was designated as high, the NOAEL would need to decrease by more than an order of magnitude to result in and HQ greater than 1. Therefore, BEHP is likely to pose low risk to spotted sandpiper

A.7.3.3.2 Great blue heron

Great blue heron was chosen as an ROC to represent primarily piscivorous birds in the LDW such as loons, western grebe, mergansers, double-crested cormorant, pigeon

[123] Benthic invertebrate prey should also be analyzed for DDT. Because DDT was not analyzed in the amphipod tissue data evaluated as part of Phase 1, analysis of this pair was not possible.

guillemot, Caspian tern, osprey, and common murre. Results for the risk characterization for great blue heron are summarized in Table A-7-38.

Table A-7-38. Summary of risk characterization for great blue heron

COPC	NOAEL HQ	LOAEL HQ	No. of TRV Studies	Uncertainty in TRV	Exposure Data
Lead	0.04	0.004	5[a]	medium	wb shiner perch (n=3, 10 fish/composite)
PCBs - dietary	0.27	0.12	6	medium	wb shiner perch (n=3, 10 fish/composite)
PCBs - eggs	**6.6**	2.9	4	medium	heron eggs (n=5, 1 egg/sample)[b]
PCB TEQs - eggs	**3.6**	1.8	8	medium	heron eggs (n=5, 1 egg/sample)[b]
Mercury	**1.7**	0.17	6	medium	wb shiner perch (n=3, 10 fish/composite)

Note: HQs greater than 1.0 are noted in **bold type**.

wb – whole-body

[a] not including three studies with chicken or quail egg production endpoint

[b] for two of the five samples, data are only available for well-developed embryo and remaining yolk sac samples, not whole-egg samples.

LOEC-based HQs for PCBs were greater than 1 (2.9) based on available heron egg data, but were less than 1 (0.12) based on a dietary approach. Thus, based on available data, exposure of great blue heron to PCBs in the LDW area may be near the level where adverse effects on reproduction could occur. Key uncertainties in the heron/PCB pair include great blue heron tissue data for prey species (assumed to be primarily fish), prey fish species preferences, and site use of herons from the nearby colony where eggs were collected. Based on these results and uncertainties, collection of additional prey tissue data is recommended for Phase 2 to reduce these uncertainties, and further evaluation of this pair is recommended in the Phase 2 ERA.

The NOAEL-based HQ for heron/mercury was also greater than 1 (1.7). Because exposure estimates for this pair were also based on limited prey fish tissue data, collection of additional prey tissue data[124] is recommended for use in further evaluation of this pair in Phase 2.

The lead NOAEL-based HQ for great blue heron was substantially less than 1 (0.041). Although tissue data were limited, it is unlikely a more robust dataset would increase the HQ to 1; prey tissue concentrations would need to be at least 45 mg/kg dw compared to maximum measured concentration in fish of 0.88 mg/kg dw. Therefore, lead is likely to pose a low risk to heron, and collection of additional data to reduce uncertainties in this pair is unlikely to change this risk conclusion.

[124] Fish collected to reduce uncertainties in heron exposure estimates should also be analyzed for DDT to reduce uncertainties in the heron/DDT pair evaluated in the problem formulation.

A.7.3.3.3 Bald eagle

Bald eagle was chosen as an ROC to represent piscivorous birds such as those listed for great blue heron, in addition to carnivorous birds such as peregrine falcon. In addition to fish, both eagle and falcon may also consume aquatic birds such as seabirds and waterfowl. The results of the risk characterization for bald eagle are summarized in Table A-7-39.

Table A-7-39. Summary of risk characterization for bald eagle

COPC	Highest NOAEL HQ[a]	Highest LOAEL HQ	No. of TRV Studies	Uncertainty in TRV	Exposure Data
Lead	0.02	0.002	5 [b]	medium	wb shiner perch (n=3, 10 fish/composite) wb English sole (n=3, 20 fish/composite)
Mercury	**1.1**	0.11	6	medium	wb shiner perch (n=3, 10 fish/composite) wb English sole (n=3, 20 fish/composite)
PCBs	0.29	0.13	6	medium	wb shiner perch (n=3, 10 fish/composite) wb English sole (n=3, 20 fish/composite) wb juvenile chinook salmon (n=26, 2-10 fish/composite)

Note: HQs greater than 1.0 are noted in **bold type**.

wb – whole-body

[a] NOAELs based on an SUF of 1 (risks were also calculated for an SUF of 0.25)

[b] not including three studies with chicken or quail egg production endpoint

No LOAEL-based HQs exceeded 1 for eagle based on the available data. The highest NOAEL-based HQ was for the eagle/mercury pair. The NOAEL-based HQ for mercury was 1.1, based on the assumption that eagles obtain all of their food from the LDW. Using the assumption that eagles obtain only 25% of their food from the LDW (which is more reasonable), the NOAEL-based HQ was 0.28. To reduce uncertainties in these estimates, collection of additional prey tissue data[125] is recommended for Phase 2, and further evaluation of this pair is recommended in the Phase 2 ERA. Reducing uncertainties in the eagle diet (including bird consumption) as well as site usage uncertainties are considered to be more resource-intensive (Table A-7-35).

NOAEL-based HQs for eagle/PCBs were less than 1 (0.29) based on available data. However, using literature-based fish to bird biomagnification factors, a conservative estimate of bird tissue PCB concentrations could result in HQs great than 1. Under this scenario, the NOAEL- and LOAEL-based HQs were 5.4 and 2.3, respectively,

[125] Prey items collected to reduce uncertainties in eagle exposure estimates should also be analyzed for DDT to reduce uncertainties in the eagle/DDT pair evaluated in the problem formulation.

assuming a SUF of 1 (see Section A.7.3.2.2). The bird tissue estimate is considered conservative because it is based on the assumption that the birds consumed by eagle eat only fish from the LDW. However, given the possibility of higher exposure in eagles from bird consumption, it is recommended that additional analysis of the eagle/PCB pair be conducted in Phase 2.

Risk to eagle from lead exposure is considered to be low based on available data. The lead NOAEL-based HQ was very low (0.021) even with the conservative assumption that eagles feed only in the LDW (i.e., SUF of 1). Lead prey concentrations would need to increase from approximately 0.9 mg/kg dw to 70 mg/kg dw for the NOAEL-based HQ to reach 1. Therefore, collection of additional data to reduce uncertainties in the eagle/lead pair is unlikely to change this risk conclusion in Phase 2.

A.7.3.3.4 River otter

Risk was characterized for river otter to represent the most highly exposed semi-aquatic mammal using the LDW. Results from the risk characterization for river otter are summarized in Table A-7-40.

Table A-7-40. Summary of risk characterization for river otter

COPC	NOAEL HQ	LOAEL HQ	No. of TRV Studies	Uncertainty in TRV	Exposure Data
PCBs	**8.5**	0.85	9	low	wb shiner perch (n=3, 10 fish/composite) wb English sole (n=3, 20 fish/composite) wb juvenile chinook salmon (n=26, 2–10 fish/composite) mussels (n=22, 50–100 mussels/composite) crab edible meat (n=2,1–3 crabs/composite) crab hepatopancreas (n=1, 3 crabs/composite)
Arsenic	**6.1**	0.61	3	high [a]	wb shiner perch (n=3, 10 fish/composite) wb English sole (n=3, 20 fish/composite) mussels (n=22, 50–100 mussels/composite) crab edible meat (n=2, 3 crabs/composite) crab hepatopancreas (n=1, 3 crabs/composite)
Lead	0.12	0.04	6	medium	wb shiner perch (n=3, 10 fish/composite) wb English sole (n=3, 20 fish/composite) mussels (n=22, 50–100 mussels/composite)) crab edible meat (n=2, 3 crabs/composite)) crab hepatopancreas (n=1, 3 crabs/composite))

Note: HQs greater than 1.0 are noted in **bold type**.

wb – whole-body

[a] exposure routes were via drinking water or gavage

No LOAEL-based HQs for river otter were greater than 1. However, the NOAEL-based HQs for PCBs (8.5) and arsenic (6.1) both exceeded 1, so it is recommended that these pairs be further evaluated in Phase 2. The primary uncertainty associated with the otter/PCBs pair is the small tissue dataset available to estimate otter exposure. To

reduce this uncertainty, collection of additional otter prey tissue data is recommended for Phase 2. These data would also reduce uncertainties in the otter/arsenic pair.

The NOAEL-based HQ for lead was 0.12. The primary uncertainty in the lead risk calculation was associated with the small number of fish tissue samples. Concentrations of lead would need to be about 14 mg/kg dw in all prey items for the NOAEL-based HQ to reach 1; existing data show concentrations range from 0.73 to 2.2 mg/kg dw in mussels, crab, and whole-body fish ranging in size from 25 to 33 cm. Concentrations of lead in otter prey in excess of 14 mg/kg dw is considered unlikely, so risk to river otter from lead is considered to be low. Additional data collection to reduce uncertainties in this pair is unlikely to change the risk conclusions.

A.7.3.3.5 Harbor seal

The harbor seal was chosen to represent other marine mammals in the LDW (sea lion and porpoise), although dietary exposures and site usage of California sea lions and harbor porpoises may differ (Section A.7.3.2.1). Results of the harbor seal risk characterization are summarized in Table A-7-41.

Table A-7-41. Summary of risk characterization results for harbor seal

COPC	NOAEL HQ	LOAEL HQ	No. of TRV Studies	Uncertainty in TRV	Exposure Data
PCBs	0.69	0.07	9	low	wb shiner perch (n=3, 10 fish/composite) wb sole (n=3, 20 fish/composite) wb juvenile chinook salmon (n=26, 2-10 fish/composite)

Note: HQs greater than 1.0 are noted in **bold type.**

wb – whole-body

Using an SUF of 0.33, which is assumed to be equally conservative for sea lions and harbor porpoises, the NOAEL-based HQ for PCBs was 0.69. However, due to the uncertainty associated with limited chemistry data for upper-trophic-level fish, and information on whether these upper-trophic-level fish and bottom-feeding fish constitute a significant proportion of the seal's diet, collection of additional data to reduce uncertainties for this pair is recommended for Phase 2.

A.7.3.3.6 Wildlife risk conclusion summary

In summary, based on a synthesis of risk estimation results and the uncertainty assessment for wildlife, the following recommendations were made, based on the available data. Note that the list of analytes to be measured in Phase 2 data collection will be identified in the Phase 2 work plan, and the final COPC list for the Phase 2 ERA will be determined in the Phase 2 Problem Formulation.

- **Spotted sandpiper.** Lead is recommended for further evaluation in the Phase 2 ERA. Collection of additional data for PCBs, copper, and zinc is recommended in Phase 2. Based on available data, risk from BEHP appears to be low.
- **Great blue heron.** PCBs and mercury are recommended for further evaluation in the Phase 2 ERA. Based on existing data, risks from lead appear to be low.
- **Bald eagle.** PCBs and mercury were recommended for further evaluation in the Phase 2 ERA. Based on existing data, risks from lead appear to be low.
- **River otter.** PCBs and arsenic were recommended for further evaluation in the Phase 2 ERA. Based on existing data, risks from lead appear to be low.
- **Harbor seal.** Collection of additional exposure data for PCBs is recommended in Phase 2.

A.7.4 Risk Characterization for Plants

This section presents an estimation of risk by calculating HQs for aquatic rooted plants based on information presented in Section A.6. Following the risk estimation, a detailed evaluation of uncertainty associated with these calculations is presented. Finally, this section presents a risk conclusion that integrates HQ results with associated uncertainty.

A.7.4.1 Risk estimation

Due to the relatively high level of uncertainty in both exposure and effects data for plants within the LDW, ranges of exposure and effects data are presented and corresponding ranges of HQs were calculated (Table A-7-42). HQs were also calculated using the 95% UCL on the mean concentration in marsh sediments (n=7 samples) and the NOEC or LOEC identified in Section A.6.2 as most suitable.[126]

[126] TRV selected based on weight of evidence of data as well as background considerations for Pb and Zn.

Table A-7-42. Range of HQs for rooted aquatic plant/COPC pairs

COPC	MARSH[a] SEDIMENT EXPOSURE CONCENTRATIONS (mg/kg dw); RANGE AND 95% UCL ON THE MEAN[b]	NOECs AVAILABLE, RANGE AND SELECTED TRV (mg/kg dw soil)	LOECs AVAILABLE, RANGE AND SELECTED TRV (mg/kg dw soil)	BACKGROUND CONCENTRATIONS	NOEC HQs (range and selected[c] TRV/mean conc)	LOEC HQs (range and selected TRV/mean conc)
Lead	9.3 – 330 (158)	9.0 – 5,000 (100)	21 – 30,000 (125)	0.10U – 24[d]; 29.6[e]; 20[f]	0.002 – **37** **(1.6)**	0.0003 – **16** **(1.3)**
Mercury	0.090 – 0.37 (0.25)	na[g]	na[g]	0.010 – 0.28[d]; 0.0944[e]; 0.15[f]	0.60 – **2.5** **(1.7)**[h]	0.60 – **2.5** **(1.7)**[h]
PCBs	0.020 – 9.4 (1.7)	10 – 1,000 (20)	40 – 1,000 (327)	0.0031 – 0.050U[d]; 0.047[f]	0.00002 – 0.94 (0.09)	0.00002 – 0.24 (0.005)
Zinc	56 – 155 (133)	10 – 2,500 (20)	25 – 5,000 (40)	15 – 101J[e]; 132.5[e]; 103[f]	0.02 – **16** **(6.7)**	0.01 – **6.2** **(3.3)**

Note: HQs greater than 1 are noted in **bold type**.

U – Undetected

J – Estimated

[a] Concentrations of COPC within 50 m of marsh habitat (per USFWS designation) (n=7 stations: DR013, DR014, DR061, DR263, DR264, DR270, DR271; see RI Maps 2-5a through 2-5k)

[b] Nondetects were treated as half the detection limit in the 95% UCL mean calculations

[c] Selection of a TRV for plants is discussed in Section A.6.2

[d] PTI (1991) (range of concentrations from Puget Sound sediment reference areas)

[e] Ecology (1994) (maximum concentration in Puget Sound region natural soil background)

[f] PTI (1991) (proposed Puget Sound reference area performance standard [i.e., sites with concentrations lower than these standards are suitable for reference area classification])

[g] No acceptable studies were identified for mercury and plants

[h] Proposed reference area performance standard used in place of NOEC or LOEC because no TRVs were available (i.e., HQ was based on a comparison to background concentration rather than a TRV)

HQs for lead ranged[127] from 0.002 - 37 and from 0.0003 - 16, using all available NOEC and LOEC data, respectively, as well as the range of lead measured in marsh sediment. Using the NOEC and LOEC concentrations presented in Section A.6.2 and the 95% UCL on the mean marsh sediment concentration, HQs were 1.6 and 1.3, respectively. Using the maximum lead concentrations measured in or near the marsh areas and the selected NOEC and LOEC, HQs of 3.3 and 2.6 were calculated, respectively.

No acceptable toxicity thresholds were identified in the literature for mercury. Thus, exposure concentrations in marsh areas of the LDW were compared to the proposed reference area performance standard for mercury as an indicator for background concentrations in Puget Sound sediments (PTI 1991). Mercury concentrations in or near marsh sediments were found to be similar to the performance standard (with HQs ranging from 0.6 to 2.5). Note, however, that these HQs were relative to background sediment data, and not effects data. Thus, these HQs do not reflect effects-based risk estimates, but instead are reflective of exposure concentrations relative to background.

HQs calculated for PCBs were less than 1 under all potential scenarios using available data (maximum NOEC-based HQ was 0.94).

For zinc, although concentrations measured in marsh sediments were similar to background concentrations (Table A-7-42), HQs ranged from 0.02 - 16 and 0.01 - 6.4 using NOEC and LOEC data, respectively. Using the 95% UCL on the mean concentration in marsh sediment and the TRVs selected in Section A.6.2, NOEC- and LOEC-based HQs were 6.7 and 3.3, respectively. Thus, although some locations sampled in or near marsh areas have concentrations higher than the low end of the wide range of available TRVs, concentrations measured in marsh sediment were only slightly above reference concentrations (i.e., the maximum concentration is a 1.5 times the proposed reference area performance standard designed to indicate sediments suitable as reference areas).

A.7.4.2 Uncertainty assessment

In this ERA, relative to the sections discussing other ROCs, the plant assessment is likely to have the highest level of uncertainty due to the paucity and questionable applicability of both the available exposure and effects data. However, because plants are increasingly being recognized in ERAs as important components of the ecosystem, the plant assessment was included to provide a summary of available information to evaluate potential impacts to aquatic plants from sediment-associated chemicals. This section presents specific areas of uncertainty in the plant assessment based on where these uncertainties are identified in the ERA (i.e., the problem formulation, exposure assessment, or effects assessment). The purpose of this uncertainty assessment is to indicate potential data gaps in the available knowledge regarding risk to plants in the LDW from sediment-associated chemicals, and to discuss, to the degree possible, what

[127] Ranges are presented because a wide range of TRVs was available for plants; uncertainty is thus very high.

impact these uncertainties could have on the assessment of risks to plants. A summary of uncertainties and their potential impact on the risk characterization is presented in Section A.7.4.2.4.

A7.4.2.1 Problem formulation

Two key areas of potential uncertainty in the plant assessment problem formulation are discussed in this section:

- Selection of rooted aquatic plants (versus algae or other plants)
- Use of sediment COPC concentrations as the key indicator of exposure relative to water exposure
- Additional exposure and effects uncertainties were also important components of the assessment in the problem formulation; these uncertainties are discussed in Sections A.7.4.2.2 and A.7.4.2.3.

Selection of Rooted Plants as the ROC

Three types of plants play key roles in maintaining high productivity in estuaries like the LDW: 1) phytoplankton suspended within the photic zone of the water column; 2) benthic microflora (microscopic plants) living on the sediment surface wherever sufficient light reaches the bottom; and 3) macroflora (rooted plants) and periphyton growing in shallow water and along the shoreline. The following two issues were the primary consideration in the final selection of rooted aquatic plants to represent potential impacts to plants from contaminated sediments:

- Rooted plants receive the most direct, and thus potentially highest exposure, from sediment-associated chemicals;
- The relative sensitivity of the three types of plants listed above are unknown (e.g., it is not known if benthic microflora are more or less sensitive than macroflora (rooted plants)).
- Thus, while selection of rooted plants makes sense due to the direct pathway (sediment to plant), it is uncertain whether analysis of risk to rooted plants is protective of all plants in the LDW. Additional toxicity information would be required to further resolve this issue.

Use of Sediment Data to Estimate Exposure

Carex (sedges) and *Scirpus* (bulrushes) are the predominant vegetation type between Turning Basin 3 and Kellogg Island. Downstream from Kellogg Island are more marine plants such as *Salicornia* (grassworts), *Distichlis* (salt grass), and *Atriplex* (salt bush). The naturally occurring *Carex* patches surveyed in 1993 occurred between elevations of 5.2 to 9.7 ft MLLW, and the single patch of naturally occurring *Scirpus* was at 12 ft (Cordell 2001a). Thus, these plants are seldom present under water. Because these predominant marsh plants are rarely underwater, use of sediment/soil exposure data were deemed to be more appropriate than using water exposure data. In addition, effects data for rooted

plants are available in soils, thus use of sediment, instead of water data were more relevant.

A7.4.2.2 Exposure assessment

This section discusses the two key uncertainties identified for plant exposure: use of marsh data and potential implications of future restoration projects.

Use of Marsh Data

Although marsh and intertidal sediment data were used to assess potential exposure to rooted plants in the LDW (see Sections A.2.4.8.2 and A.6.11), marsh sediment data were emphasized. Tidal elevation and salinity gradients are the main determinants of the potential distribution for estuarine rooted plants. The most productive areas for estuarine plant communities are found in tidal marshes. Marsh soils are generally fine-textured and nutrient-rich, and support grasses, sedges, rushes, and various other types of plants associated with maritime and estuarine habitats. In the LDW, there is a total of 1.75 ha of habitat for macrophytes, primarily limited to portions of Kellogg Island and other small intertidal areas with vegetated intertidal habitat (USFWS 2000).

Because the number of sediment samples from marsh areas was small (n=7), there is some uncertainty as to how representative these data are with respect to plant growth areas. In addition, there is some question as to whether plants may grow in intertidal areas that have not been identified as marsh habitat. Because the 95% UCL on the mean concentrations for lead, PCBs, and zinc were 2.3, 4.6, and 2.1 times higher (respectively) in the intertidal than in marsh areas (mercury concentrations were similar), it is possible that rooted plants may be exposed to areas with higher concentrations than those reported for marsh areas. However, because NOECs and LOECs ranged over two orders of magnitude for lead, PCBs, and zinc (Table A-6-3), the ability to interpret risk from small differences in sediment concentrations is limited. In addition, much of the intertidal area in the LDW would not be conducive to plant growth due to habitat constraints, such as rip rap.

Potential Implications of Future Restoration Projects

Through the efforts of future restoration projects, it is possible that additional marsh or intertidal habitat may be created that would be conducive to plant growth. If these areas contained elevated concentrations of COPCs, it is possible that plants may grow in areas with greater COPC exposure. However, interpretation of risk from this exposure would be difficult to assess due to the high uncertainty in the effects literature (see Section A.7.4.2.3).

A7.4.2.3 Effects assessment

The effects data for plants used in this ERA are highly uncertain. Four general areas of uncertainty are discussed in this section:

- The applicability of soil toxicity data relative to sediment exposures

- The relevance of plants such as corn, oats, wheat, and soybeans relative to the sedges, bulrushes, grassworts, salt grasses, and salt bush of the LDW
- The uncertainty in NOECs and LOECs found in the literature of the soil-based toxicity data for these agricultural plants
- The lack of mercury toxicity literature

As part of the problem formulation, the literature was searched for toxicity data for rooted plants in aquatic systems. No data were located to relate concentrations in sediment to potential adverse impacts on rooted plants. Thus, concentrations in soil were used instead (based on Efroymson et al. [1997], a comprehensive review of soil toxicity literature). No studies have been conducted to determine applicability of these values to sediment exposure of aquatic rooted plants, and thus their relevance is unknown.

Furthermore, the soil toxicity literature is based largely on agricultural species (e.g., wheat, soybeans, corn). The sensitivity of these plants relative to those that grow in habitat present in the LDW is unknown. Even the toxicity data available for these species is widely variable with NOECs and LOECs reported that range over two orders of magnitude, depending in part on the endpoint and exposure conditions (Table A-6-3).

For mercury, no toxicity data were available from a controlled laboratory study. The only study identified (Panda et al. 1992) assessed toxicity in plants in a sludge from a chloralkali plant with elevated mercury concentrations. However, because a NOEC of 35 mg/kg mercury was reported for barley plant height and seed germination, it seems unlikely that phytotoxicity from mercury would occur in the LDW, which had a maximum concentration of 4.5 mg/kg Hg in LDW sediment site wide.

A.7.4.2.4 Summary of uncertainties for plants

Summaries of uncertainties in the plant assessment are presented in Tables A-7-43 and A-7-43. As discussed above, the key uncertainty for plants is the general inadequacy of available effects data, particularly for lead, which had concentrations in marsh sediments greater than background concentrations.

Table A-7-43. Summary of primary uncertainties in plant risk characterization

Issue	Level of Uncertainty	Effect of Uncertainty on Risk Estimate	Potential Means to Decrease Uncertainty	Potential Impact on Risk Conclusions	Feasibility to Fill Data Gap
Exposure Assessment					
Use of sediment data alone to estimate exposure	Medium	Possibly underestimated	Estimate exposure through water	Low	Medium
Use of limited marsh dataset	Medium	Accuracy unknown	Collect additional sediment data	Medium	High
Impact of potential restoration projects	Medium	Overestimated in areas to be restored	Assess during planning of potential projects	Low	Low
Effects Assessment					
Soil vs. sediment toxicity data	High	Accuracy unknown	Develop alternative TRVs	Medium	Low
Relevance of plants studied in literature	Medium	Accuracy unknown	Develop alternative TRVs	Medium	Low
Large range of NOECs and LOECs reported in literature	High	Possibly overestimated	Assess risks using median TRV	High	Medium

Level of uncertainty key: **low** = large or relevant dataset
medium = small dataset or limited information
high = very limited data

Potential impact key: **low** = unlikely to result in a change of HQ from less than 1 to greater than 1 (or vice versa)
medium = could result in a change of HQ from less than 1 to greater than 1 if worst-case scenario is used (scenario is viewed as unlikely)
high = HQ could change from less than 1 to greater than 1 (or vice versa) using a scenario that is conservative but more reasonable than the worst-case scenario

Feasibility key: **low** = high budget or difficult research study would be required to address uncertainty
medium = issue could be resolved with a mid-level field sampling event or research study or a detailed assessment of literature
high = issue could be resolved with additional literature search or through limited field sampling

Table A-7-44. Summary of uncertainties in TRVs used in plant risk characterization

TRV	Level of Uncertainty	Potential Impact on Risk Conclusions
Lead	high	high
Zinc	high	high
PCBs	high	low [a]
Mercury	high	medium [b]

[a] Entire range of TRVs resulted in HQs less than 1.

[b] TRV based on field study with mercury and co-located contaminants was much higher than site-wide mercury concentrations.

A.7.4.3 Risk conclusions

This section integrates the results of the risk estimation with the uncertainty assessment to describe the level of risk estimated for rooted plants in the LDW.

Scenarios were possible for NOEC- and LOEC-based HQs to be greater than 1 for zinc and lead. However, because zinc concentrations in and near marsh sediment were also similar to background concentrations (55-155 mg/kg in marsh sediments vs. 15-133 mg/kg background indicators), potential exposure of plants to zinc appeared to be similar to that of reference conditions in the Puget Sound. Thus, risk to plants from zinc in the LDW appears to be low (Table A-7-45).

Concentrations of lead in or near marsh sediments were higher than background levels. NOEC- and LOEC-based HQs ranged from 0.0019 to 37 and 0.0003 to 16, respectively. Due to the large range and the high uncertainty in the effects data, it is highly uncertain if rooted aquatic plants in the LDW are at risk from lead. Because uncertainty also exists in lead exposure in marsh areas, the potential to collect additional sediment data in these areas will be discussed in the data gaps memorandum. However, due to the high uncertainty in the effects data, these data are unlikely to change the risk conclusions.

PCBs and mercury are believed to pose low risk to plants in the LDW. PCB concentrations in and near marsh sediments are lower than the range of TRVs available for plants. No TRVs are available for mercury because the only study available (Panda et al. 1992) contained co-located contaminants. However, risks are believed to be low for mercury because 1) the maximum concentration of mercury in LDW sediment (sitewide) was an order of magnitude less than the NOEC in Panda et al. (1992); and 2) concentrations of mercury in or near marsh areas were similar to background levels.

Table A-7-45. Summary of risk characterization for plants

COPC	RESULT	COMMENT
Lead	risk possibility elevated	HQs can exceed 1, but large uncertainties in effects data
Mercury	low risk	No mercury-specific TRV available; concentrations near background
PCBs	low risk	HQs < 1 for all scenarios
Zinc	low risk	HQs can exceed 1, but concentrations in marsh sediments near background and large uncertainty in effects data

A.8 Conclusions

Using sediment and tissue chemistry data collected within the last 10 years, the Phase 1 ERA evaluated risks from sediment-associated chemicals to benthic invertebrates, crab, fish and wildlife species that may reside or forage in the LDW for at least a portion of their lives. Although there is relatively little suitable habitat presently available for rooted aquatic plants within the LDW, risks to this group were also evaluated. Based on risk estimates and assessments of uncertainty, ROC/COPC pairs were discussed, and critical data needs were identified, as summarized below.

Additional site-specific data will be collected in Phase 2 to fill data gaps and to reduce uncertainties identified in the Phase 1 ERA. These data, and the results of the Phase 1 ERA, will be evaluated as part of the Phase 2 ERA. The Phase 1 ERA also provided information used in the identification of candidate sites for early remedial action.

A.8.1 Benthic Invertebrates

Locations where potential adverse effects to benthic invertebrates were predicted were identified by comparing existing surface sediment chemistry data to SMS numeric standards and DMMP guidelines. Based on this analysis and available data, approximately 70% of the LDW was predicted to pose low risk to benthic invertebrates because of the lack of SQS/SL exceedances for any chemicals. There is greater likelihood of adverse risks to benthic invertebrates in the 10% of the area in the LDW with exceedances of CSLs or MLs for at least a single chemical. The area between these two categories (approximately 20%) can be categorized as having an intermediate likelihood of adverse effects because COPC concentrations in these areas were above the SQS/SL but below the CSL/ML. Potential adverse effects addressed by the existing standards included mortality and abnormal development at the individual level and altered ecological function at the community level. All COPCs for benthic invertebrates will be further evaluated in the data gaps process and the Phase 2 ERA. To evaluate potential impacts to benthic invertebrates from TBT exposure, a range of HQs was calculated using either measured maximum tissue concentrations, or tissue concentrations estimated using a modified BSAF and minimum, median, or maximum TBT sediment concentrations. Due to the potentially high HQ (up to 5.3 using the maximum sediment concentration in the LDW to estimate the tissue concentration), TBT will be further evaluated in the data gaps process and the Phase 2 ERA.

Risk to crabs from exposure to sediment-associated COPCs in the LDW appeared to be relatively low based on the limited data available on exposure and effects, with the possible exception of arsenic, which had a NOEC-based HQ of 10. However, because this HQ was based only on a NOEC (no studies were found in the literature associating a tissue arsenic concentration with an adverse effect), this HQ is highly uncertain. Collection of additional data to reduce uncertainties in the crab risk assessment will be discussed as part of the data gaps process, and risks to crab will be further evaluated in the Phase 2 ERA.

A.8.2 Fish

Based on the existing data evaluated in Phase 1, the only ROC/COPC pairs with LOEC-based HQs greater than 1 for fish species were:

- English sole/copper, which had a LOEC-based HQ of 7.6 for the growth endpoint (the NOEC-based HQ was 15)
- Bull trout/PCBs, which had a LOEC-based HQ of 2.1 for the growth endpoint (the NOEC-based HQ was 8.2)

- English sole/arsenic, which had a LOEC-based HQ of 1.1 for the growth endpoint (the NOEC-based HQ was 1.6)

Based on these preliminary risk estimates, copper, arsenic, and PCBs in LDW sediments may result in adverse impacts to certain fish species because preliminary exposure estimates using existing data indicated exposure concentrations greater than the lowest relevant effects data. However, because average arsenic concentrations were not highly elevated in LDW sediments relative to background concentrations of this metal in central Puget Sound sediments, issues related to regional background levels will be discussed further in Phase 2 based on EPA (2002) guidance.

The interpretation of NOEC-based HQs is less certain than LOEC-based HQs because the true threshold for effect is somewhere between the two TRVs. Table A-8-1 provides a summary of the 11 ROC/COPC pairs with NOEC-based HQs greater than 1. Collection of additional data to reduce uncertainties in the fish risk assessment will be discussed as part of the data gaps process, and risks to fish will be further evaluated in the Phase 2 ERA.

Table A-8-1. Summary of ROC/COPC pairs with NOEC-based HQs greater than 1

ROC/COPC Pair	NOEC-based HQ	Endpoint	LOEC-based HQ
English sole/copper	15	growth	7.6
Bull trout/PCBs	8.2	growth	2.2
English sole/arsenic	1.6	growth	1.1
Bull trout/mercury	2.2	survival	0.94
Bull trout/PCBs	4.2	reproduction	0.87
English sole/PCBs	2.4	growth	0.62
English sole/PCBs	1.2	reproduction	0.25
Bull trout/mercury	2.1	reproduction	0.21
Juvenile chinook salmon/PAHs	1.7	growth	0.17
Bull trout/TBT	1.1	survival	0.11
Juvenile chinook salmon/TBT	1.1	survival	0.11

A.8.3 Wildlife

None of the preliminary exposure estimates for wildlife ROCs resulted in dietary doses greater than those associated with adverse effects, based on existing data. However, heron egg data, an additional line of evidence, indicated risks from PCBs may be higher than those estimated using the dietary approach. In addition to PCBs, NOAEL-based HQs for arsenic, lead, and mercury were greater than 1 (Table A-8-2). Note, however, that interpretation of NOAEL-based HQs is less certain than LOAEL-based HQs because the true threshold for effect is somewhere between the two TRVs. Overall, Phase 1 risk estimates based on dietary assessments appear to be low or negligible for wildlife species that use the LDW. However, the heron PCB egg data indicated a potential risk to herons. Collection of additional data to reduce uncertainties in the wildlife risk assessment will

be discussed as part of the data gaps process, and risks to wildlife will be further evaluated in the Phase 2 ERA.

Table A-8-2. Summary of ROC/COPC pairs with NOEC-based HQs greater than 1

ROC/COPC Pair	NOAEL-based HQ	Endpoint	LOAEL-based HQ
Great blue heron/PCBs (based on total PCBs in eggs)	6.6	reproduction	2.9
River otter/PCBs	8.5	reproduction	0.85
River otter/arsenic	6.1	reproduction	0.61
Sandpiper/lead	4.1	reproduction	0.41
Great blue heron/mercury	1.7	reproduction	0.17
Bald eagle/mercury (SUF = 1)	1.1	reproduction	0.11

A.8.4 Plants

The preliminary risk estimates for plants were much more uncertain than the other assessments in this Phase 1 ERA. Both exposure and effects data were highly uncertain. Of the four COPCs evaluated for plants, risk estimates from sediment-associated zinc appear to be the greatest, but concentrations of zinc in marsh sediment were within the range of background levels (Table A-7-45). Lead concentrations in marsh sediment were greater than background sediment concentrations, and could result in adverse effects under a worst-case scenario. However, if the range of sediment concentrations in marsh areas and the range of effects data are considered, the likelihood of risks to plants from sediment-associated lead in marsh areas is relatively low. The potential for additional fieldwork to reduce uncertainties in the risk estimates for lead will be discussed in the data gaps memorandum, but collection of these data is unlikely to change the risk conclusions.

A.8.5 Uncertainties

There are a number of uncertainties associated with the risk estimates presented in this assessment. Due to the conservative nature of many of the assumptions used in this scoping-phase assessment, risks were likely overestimated for many of the ROC/COPC pairs. However, due to limited site-specific data, it is possible some risks could have been underestimated. The collection of additional data or performance of additional analyses as part of the Phase 2 ERA should reduce many of the uncertainties identified. Depending on the direction and magnitude of the uncertainty, additional data could result in identification of additional COPCs or eliminate COPCs currently identified in this Phase 1 preliminary risk characterization.

A.8.6 Next Steps

The risk estimates presented in this ERA for some of the ecological receptors exceeded levels identified by SMS and ecological risk guidance, and thus suggest that remedial

action may be warranted in some areas of the LDW. It is likely that early remedial actions undertaken within in the LDW will reduce this risk significantly. The Phase 2 ERA will include an analysis to evaluate the impact of these early actions on residual risk. Identifying the extent of remediation to address residual risk as part of Phase 2 may require that a linkage between sediment and tissue concentrations be derived or assumed. It is likely that some type of quantitative modeling of this linkage will be performed as part of the Phase 2 RI.

Although risk estimates to fish, wildlife, and plants did not reach the level required to trigger the identification of additional areas in the LDW for early action,[128] these receptors will benefit from any remedial action that results in lower concentrations of chemicals in surface sediments, particularly those chemicals discussed below. Results from the benthic invertebrate ERA were used directly in the identification process, and risks to these species will also be reduced through early remedial action.

A.9 References

ACOE. 2002. The Environmental Residue-Effects Database (ERED). http://www.wes.army.mwil/el/ered/. Accessed January 16, 2002.

Adams JA, Conard B, Ethier G, Brix KV, Paquin RP, DiToro DM. 2000. The challenges of hazard identification and classification of insoluble metals and metal substances for the aquatic environment. Hum Ecol Risk Assess 6(6):019-1038.

Aery NC, Sakar S. 1991. Studies on the effect of heavy metal stress on growth parameters of soybean. J Environ Biol 12(1):15-24.

Ahlborg UG, Becking GC, Birnbaum LS, Brouwer A, Derks HJGM, Feeley M, Golor G, Hanberg A, Larsen JC, Liem AKD, Safe SH, Schlatter C, Waern F, Younes M, Yrjanheikki E. 1994. Toxic equivalency factors for dioxin-like PCBs. Chemosphere 28:1049-1067.

Alexander G. 1977. Food of vertebrate predators on trout waters in north central lower Michigan. Mich Academic 10:181-195.

Allison DT, Kollman BJ, Cope OB, Van Valin C. 1964. Some chronic effects of DDT on cutthroat trout. Research report 64. Bureau of Sport Fisheries and Wildlife, US Fish and Wildlife Service, Washington, DC.

Allen JR, Barsotti DA, Carstens LA. 1980. Residual effects of polychlorinated biphenyls on adult nonhuman primates and their offspring. J Toxicol Environ Health 6:55-66.

Ambrose AM, Larson PS, Borzelleca JF, Hennigar GR Jr., Verrett J. 1976. Long term toxicologic assessment of nickel in rats and dogs. J Food Sci Technol 13:181-187.

Amend DF. 1981. Potency testing of fish vaccines. Dev Biol Stand 49:447-454.

[128] For a fish or wildlife ROC/COPC pair to trigger site identification, the LOEC- or LOAEL-based HQ has to exceed 10 (Windward 2002).

Ames J, Phinney DE. 1977. Puget Sound summer-fall chinook methodology: escapement estimates and goals, run size forecasts, and in season run size updates. Technical report. Washington Department of Fisheries, Olympia, WA.

Amrhein JF, Stow CA, Wible C. 1999. Whole-fish versus filet polychlorinated-biphenyl concentrations: an analysis using classification and regression tree models. Environ Toxicol Chem 18(8):1817-1823.

Anderson B. 2002. Personal communication (telephone conversation on 3/22/02 with Berit Bergquist, Windward Environmental LLC regarding raptors in the LDW). Falcon Research Group, Bow, WA.

Anderson JW, Rossi SS, Tukey RH, Vu T, Quattrochi LC. 1995. A biomarker, 450 RGS, for assessing the potential toxicity of organic compounds in environmental samples. Environ Toxicol Chem 14:1159-1169.

Angell CL, Miller BS, Wellings SR. 1975. Epizootiology of tumors in a population of juvenile English sole (*Parophrys vetulus*) from Puget Sound, Washington. J Fish Res Board Can 32:1723-1732.

Ankley GT, Schubauer-Berigan MK, Dierkes JR. 1991. Predicting the toxicity of bulk sediments to aquatic organisms with aqueous test fractions: Pore water vs. elutriate. Environ Tox Chem 10:1359-1366.

Arkoosh MR, Casillas E, Clemons E, McCain B, Varanasi U. 1991. Suppression of immunological memory in juvenile chinook salmon (*Oncorhynchus tshawytscha*) from an urban estuary. Fish Shellfish Immunol 1:261-277.

Arkoosh MR, Clemons E, Myers M, Casillas E. 1994. Suppression of B-cell mediated immunity in juvenile chinook salmon (*Oncorhynchus tshawytscha*) after exposure to either a polycyclic aromatic hydrocarbon or to polychlorinated biphenyls. Immunopharm Immunotoxicol 16(2):293-314.

Arkoosh MR, Casillas E, Clemons E, Kagley AN, Olson R, Reno P, Stein JE. 1998a. Effect of pollution on fish diseases: potential impacts on salmonoid populations. J Aquat Animal Health 10:182-190.

Arkoosh MR, Casillas E, Collier TK, Krahn MM, Stein JE. 1998b. Hylebos fish injury round II part 1: Effects of chemical contaminants from the Hylebos Waterway on disease resistance of juvenile chinook salmon. Northwest Fisheries Science Center, National Marine Fisheries Service, NMFS, National Oceanic and Atmospheric Administration, Seattle, WA.

Arkoosh, MR, Casillas E, Huffman P, Clemons E, Evered J, Stein JE, Varanasi U. 1998c. Increased susceptibility of juvenile chinook salmon (*Oncorhynchus tshawytscha*) from a contaminated estuary to the pathogen *Vibrio anguillarum*. Trans Am Fisheries Soc 127:360-374.

Armstrong, FAJ. 1979. Effects of mercury compounds on fish. In: Nriagu JO, ed, The biogeochemistry of mercury in the environment. Elsevier/North-Holland Biomedical Press, New York, NY. pp. 657-670.

Arizona Game & Fish. 2002. Great egret (*Casmerodius albus*). Arizona Game & Fish Department, Phoenix, AZ. Accessed 12/10/02. http://www.gf.state.az.us/frames/fishwild/ngame_f.htm.

Armstrong RH. 1996. Alaska's fish. A guide to selected species. Alaska Northwest Books, Portland, OR.

ATSDR. 1997. Toxicological profile for cyanide. Prepared for the Agency for Toxic Substances and Disease Registry, US Public Health Service, Atlanta, GA.

Aulerich RJ, Ringer RK. 1977. Current status of PCB toxicity to mink, and effect on their reproduction. Arch. Environ Contam Toxicol 6:279–292.

Aulerich RJ, Bursian SJ, Breslin WJ, Olson BA, Ringer RK. 1985. Toxicological manifestations of 2,4,5,2',4',5'-,2,3,6,2',3',6'-,and 3,4,5,3',4',5'- hexachlorobiphenyl and Aroclor 1254 in mink. J Toxicol Environ Health 15:63-79.

Ax RL, Hansen LG. 1975. Effects of purified polychlorinated biphenyl analogs on chicken reproduction. Poult Sci 54:895–900.

Azar A, Trochimowicz HJ, Maxfield ME. 1973. Review of lead studies in animals carried out at Haskell Laboratory — two-year feeding study and response to hemorrhage study. In: Barth D et al., eds, Environmental Health Aspects of Lead, Proceedings, International Symposium. Commission of European Communities, Amsterdam, pp 199-210.

Babich H, Stotzky G. 1985. A microbial assay for determining the influence of physicochemical environmental factors on the toxicity of organics: Phenol. Arch Environ Contam Toxicol 14:409-415.

Bailey SK, Davies IM. 1991. Continuing impact of TBT, previously used in mariculture, on dog whelk (*Nucella lapillus* L.) populations in a Scottish sea loch. Mar Environ Res 32:187-199.

Baker RTM, Handy RD, Davies SJ, Snook JC. 1998. Chronic dietary exposure to copper affects frowth, tissue lipid peroxidation, and metal composition of the grey mullet *Chelon labrosus*. Marine Environ Res 45:357-365.

Bane G, Robinson M. 1970. Studies on the shiner perch, *Cymatogaster aggregata* Gibbons, in upper Newport Bay, California. Wasmann J Biol 28(2):259-268.

Barry JP, Yoklavich MM, Cailliet GM, Ambrose DA, Antrim BS. 1996. Trophic ecology of the dominant fishes in Elkhorn Slough, California, 1974-1980. Estuaries 19(1):115-138

Barsotti DA, Marlar RJ, andAllen JR. 1976. Reproductive dysfunction in rhesus monkeys exposed to low levels of polychlorinated biphenyls (Aroclor 1248). Food Cosmet Toxicol 14:99-103.

Battelle. 1996. Final report for the PCB Aroclor and congener analyses on fish tissue samples from the Elliott Bay/Duwamish River project. Pacific Northwest Laboratory, Battelle Marine Research Laboratory, Sequim, WA.

Battelle, Pentec, Striplin, Shapiro, KCDNR. 2001. Reconnaissance assessment of the state of the nearshore ecosystem: Eastern shore of central Puget Sound, including Vashon and Maury Islands (WRIAs 8 and 9). Prepared for King County Department of Natural Resources, Seattle, WA. Battelle Marine Research Laboratory, Sequim, WA, with Pentec Environmental, Striplin Environmental Associates, Shapiro Associates, Inc., and King County Department of Natural Resources.

Battershill JM. 1994. Review of the safety assessment of polychlorinated biphenyls (PCBs) with particular reference to reproductive toxicity. Hum Exp Toxicol 13(9):581–597.

Bauer B, Fioroni P, Schulte-Oehlmann U, Oehlmann J, Kalbfus W. 1997. The use of *Littorina littorea* for tributyltin (TBT) effect monitoring—results from the German TBT study 1994/1995 and laboratory experiments. Env Poll 96(3):299.

Baumann PC, Smith IR, Metcalfe CD. 1996. Linkages between chemical contaminants and tumors in benthic Great Lakes fish. J Great Lakes Res 22(2):131-152.

Beach RA, Geiger C, Jeffries SJ, Treacy SD, Troutman BL. 1985. Marine mammals and their interactions with fisheries of the Columbia River and adjacent waters, 1980-1982. Third annual report. Wildlife Management Division, Washington Department of Wildlife, Olympia, WA.

Bechtel Jacobs. 1998. Biota sediment accumulation factors for invertebrates: Review and recommendations for the Oak Ridge Reservation. Prepared for US Department of Energy, Office of Environmental Management. Bechtel Jacobs Company LLC, Oak Ridge, TN. 52 pp. http://www.hsrd.ornl.gov/ecorisk/bjcor-112a1.pdf.

Becker CD. 1967. The Green River Hatchery, Washington: a historical and statistical review. Circ 67-1. Fisheries Research Institute, University of Washington, Seattle, WA (as cited in Grette and Salo [1986]).

Beckvar N, Field J, Salazar S, Hoff R. 1996. Contaminants in aquatic habitats at hazardous waste sites: mercury. NOAA Technical Memorandum NOS ORCA 100. National Oceanic and Atmospheric Administration, Seattle, WA.

Bent AC. 1929. Life histories of North American shore birds; order Limicolae. US National Museum. Bulletin, US Government Printing Office, Washington, DC.

Benton MJ, Mimrod AC, Benson WH. 1994. Evaluation of growth and energy storage as biological markers of DDT exposure in sailfish mollies. Ecotoxicol Environ Saf 29:1-12.

Bernhardt JC, Yake WE. 1981. The impact of Renton wastewater treatment plant on water quality of the lower Green/Duwamish River. Part 1 and 2. Department of Ecology, Olympia, WA.

Berntssen MHG, Hylland K, Bonga SEW, Maage A. 1999a. Toxic levels of dietary copper in Atlantic salmon (*Salmo salar* L.) parr. Aquat Toxicol 46:87-99.

Berntssen MHG, Lundebye AK, Maage A. 1999b. Effects of elevated dietary copper concentrations on growth, feed utilization and nutritional status of Atlantic salmon (*Salmo salar* L.) fry. Aquaculture 174:167-181.

Bianchini A, Gilles R. 1996. Toxicity and accumulation of mercury in three species of crabs with different osmoregulatory capacities. Bull Environ Contam Toxicol 57:91-98.

Bills TD, Marking LL, Olson LE. 1977. Effects of residues of the polychlorinated biphenyl Aroclor 1254 on the sensitivity of rainbow trout to selected environmental contaminants. Progr Fish Cult 39(3):150.

Bills TD, Marking LL, Mauck WL. 1981. Polychlorinated biphenyl (Aroclor 1254) residues in rainbow trout: effects on sensitivity to nine fishery chemicals. N Amer J Fish Manage 1:200-203.

Bingham CR. 1978. Aquatic disposal field investigations, Duwamish Waterway, disposal site Puget Sound, Washington. Appendix G: Benthic community structural changes resulting from dredged material disposal, Elliott Bay disposal site. Technical report D-77-24. U. S. Army Engineer Waterways Experiment Station, Vicksburg, MS.

Birge WJ, Black JA, Westerman AG, Francis PC, Hudson JE. 1977. Embryopathic effects of waterborne and sediment-accumulated cadmium, mercury and zinc on reproduction and survival of fish and amphibian populations in Kentucky. Research Report #100. US Department of the Interior, Washington, DC.

Birge WJ, Black JA, Westerman AG, Hudson JE. 1979. The effects of mercury on reproduction of fish and amphibians. In: Nriagu JO, ed, The biogeochemistry of mercury in the environment. Elsevier/North-Holland Biomedical Press, Amsterdam, pp 629-655.

Blazer VS, Fournie JW, Weeks-Perkins BA. 1997. Macrophage aggregates: biomarker for immune function in fishes? In: Dwyer FJ, Doane TR, Hinman ML, eds, Environmental toxicology and risk assessment: Modeling and risk assessment. Vol 6. STP 1317. American Society for Testing and Materials, Philadelphia, PA, pp 360-375.

Bleavins MR, Aulerich RJ, Ringer RK. 1980. Polychlorinated biphenyls (Aroclors 1016 and 1242): Effects on survival and reproduction in mink and ferrets. Arch Environ Contam Toxicol 9:627-635.

Blomberg G, Simenstad C, Hickey P. 1988. Changes in Duwamish River estuary habitat over the past 125 years. In: Proceedings of the First Annual Meeting on Puget Sound Research, Seattle, WA.

Bolger M. 1993. Overview of PCB toxicology. In: Proceedings, US Environmental Protection Agency's National Technical Workshop, PCBs in Fish Tissue. EPA/823-R-93-003. Office of Water, US Environmental Protection Agency, Washington, DC, pp 1-37 to 31-53.

Boothe P. 1967. The food and feeding habits of four species of San Francisco Bay fish. California Fish and Game. MRO Ref. Ser (67-13):Amer 1-151.

Boulva J, Mclaren IA. 1979. Biology of the harbor seal, *Phoca vitulina*, in eastern Canada. Fish Res Board Can Bull 200.

Braun BM, Norstrom RJ. 1989. Dynamics of organochlorine compounds in herring gulls: III. Tissue distribution and bioaccumulation in Lake Ontario gulls. Environ Toxicol Chem 8:957-968.

Brecken-Folse JA, Mayer FL, Pedigo LE, Marking LL. 1994. Acute toxicity of 4-nitrophenol, 2,4-dinitrophenol, terbufos and trichlorfon to grass shrimp (*Palaemontes* spp) and sheepshead minnows (*Cyprinodon variegates*) as affected by salinity and temperature. Environ Toxicol Chem 13: 67-77.

Britton WM, Huston TM. 1972. Yolk content and hatchability of egg from hens fed Aroclor 1242. Poult Sci 51(5):1869.

Britton WM, Huston TM. 1973. Influence of polychlorinated biphenyls in the laying hen. Poult Sci 52:1620–1624.

Brown RF, Mate BR. 1983. Abundance, movements, and feeding habits of harbor seals, *Phoca vitulina*, at Nearts and Tillamook Bays, Oregon. US Natl Marine Fish Serv Fish Bull 81:291-301.

Brunström B. 1990. Mono-ortho-chlorinated chlorobiphenyls: Toxicity and induction of 7-ethoxyresorufin O-deethylase (EROD) activity in chick embryos. Arch Toxicol 64:188-192.

Brunström B, Darnerud PO. 1983. Toxicity and distribution in chick embryos of 3,3',4,4'-tetrachlorobiphenyl injected into the eggs. Toxicology 27:103-110.

Bryan GW, Gibbs PE, Burt GR, Hummerstone LG. 1987. The effects of tributyltin (TBT) accumulation on adult dogwhelks, *Nucella lapillus*: long-term field and laboratory experiments. J Mar Biol Assoc UK 67:525-544.

Buehler DA. 2000. Bald eagle (*Haliaeetus leucocephalus*). In: Poole A, Stettenheim P, Gill F, eds, The birds of North America. No. 506. The Academy of Natural Science of Philadelphia, Philadelphia, PA.

Buhler DR, Rasmusson ME, Shanks WE. 1969. Chronic oral DDT toxicity in juvenile coho and chinook salmon. Toxicol Appl Pharmacol 14:535-555.

Burdick G, Harris EJ, Dean HJ, Walker TM, Skea J, Colby D. 1964. Accumulation of DDT in lake trout and the effect on reproduction. Trans Am Fish Soc 93:127-136.

Burke J, Fitzhugh OG. 1970. Suppl. No. 1, States report of chemistry and toxicology of PCBs. US Food and Drug Administration, Washington, DC (as cited in USAF 1989).

Burmaster DE, Thompson KM. 1997. Estimating exposure point concentrations for surface soils for use in deterministic and probabilistic risk assessments. Hum Ecolog Risk Assess 3:363-384.

Butler RW. 1992. Great blue heron. In: Poole A, Stettenheim P, Gill F, eds, The birds of North America. No. 25. The Academy of Natural Science of Philadelphia, Philadelphia, PA.

Butler RW. 1993. Time of breeding in relation to food availability of female great blue herons (*Ardea herodias*). Auk 110: 693-701.

Call DJ, Brooke LT, Lu P-Y. 1980. Uptake, elimination and metabolism of three phenols by fathead minnows. Arch Environ Contam Toxicol 9:699-714.

Canli M, Furness RW. 1995. Mercury and cadmium uptake from seawater and from food by the Norway lobster *Nephrops norvegicus*. Environ Toxicol Chem 14(5):819-828.

Canning DJ, Herman SG, Shea GB. 1979. Terminal 107 environmental studies, wildlife study. Prepared for the Port of Seattle Planning and Research Department. Oceanographic Institute of Washington and Northwest Environmental, Seattle, WA.

Carey JH. 1994. Transformation processes of contaminants in rivers. Hydrological, chemical and biological processes of transformation and transport of contaminants in aquatic environments. In: Proceedings of the Rostov-on-Don Symposium; May 1993. International Association of Hydrological Sciences (IAHS) Rostov, Russia. Pub. No 219. pp 41-50.

Carey J, Cook P, Giesy J, Hodson P, Muir D, Owens W, Solomon K. 1998. Ecotoxicological risk assessment of the chlorinated organic chemicals. SETAC Press, Pensacola, FL.

Carlson RW, Rolfe GL. 1979. Growth of rye grass and fescue as affected by lead-cadmium-fertilizer interaction. J Environ Qual 8:348-353.

Carlson RW, Bazzaz FA. 1977. Growth reduction in American sycamore (*Lantanus occidentalis* L.) caused by Pb-Cd interaction. Environ Pollut 12:243-251.

Carlson AR, Kosian PA. 1987. Toxicity of chlorinated benzenes to fathead minnows (*Pimephales promelas*). Arch Environ Contam Toxicol 16:129-135.

Casillas E, Arkoosh MR, Clemons E, Hom T, Misitano D, Collier TK, Stein JE, Varanasi U. 1995a. Chemical contaminant exposure and physiological effects in outmigrant chinook salmon from selected urban estuaries of Puget Sound, Washington. In: Keefe M, ed, Salmon Ecosystem Restoration: Myth and Reality; Proceedings of the 1994 Northeast Pacific Chinook and Coho Salmon Workshop, American Fisheries Society, Oregon Chapter, Corvallis, OR, pp. 86-102.

Casillas E, Arkoosh MR, Clemons E, Hom T, Misitano D, Collier TK, Stein JE, Varanasi U. 1995b. Chemical contaminant exposure and physiological effects in outmigrant Chinook salmon from urban estuaries of Puget Sound, Washington. Proceedings Puget Sound Research 95, Puget Sound Water Quality Authority, PO Box 40900, Olympia, WA. pp. 657-665.

Casillas E, Eberhart B-T, Collier TK, Krahn MM, Stein JE. 1998a. Hylebos fish injury study, round II, part 3: Exposure of juvenile chinook salmon to chemical contaminants specific to the Hylebos Waterway: Tissue concentrations and biochemical responses. Environmental Conservation Division, Northwest Fisheries Science Center, National Marine Fisheries Service, National Oceanic and Atmospheric Administration, Seattle, WA.

Casillas E, Eberhart B-T, Sommers FC, Collier TK, Krahn MM, Stein JE. 1998b. Hylebos fish injury study, round II, part 2: Effects of chemical contaminants from the Hylebos Waterway on growth of juvenile chinook salmon. Environmental Conservation Division, Northwest Fisheries Science Center, National Marine Fisheries Service, National Oceanic and Atmospheric Administration, Seattle, WA.

Casillas E, Misitano D, Johnson LL, Rhodes LD, Collier TK, Stein JE, McCain BB, Varansasi U. 1991. Inducibility of spawning and reproductive success of female English sole (*Parophrys vetulus*) from urban and nonurban areas of Puget Sound, Washington. Mar Environ Res 31:99-122.

Cember H, Curtis EH, Blaylock BG. 1978. Mercury bioconcentration in fish: Temperature and concentration effects. Environ Pollut 17:311-319.

Chen TT, Reid PC, Van Beneden V, Sonstegard RA. 1986. Effect of Aroclor 1254 and mirex on estradiol-induced vitellogenin production in juvenile rainbow trout (*Salmo gairdneri*). Can J Fish Aquat Sci 43:169-173.

Chliamovitch YP Kuhn C. 1977. Behavioral, haematological and histological studies on acute toxicity of bis(tri-n-butyltin)oxide on *Salmo gairdneri* Richardson and *Tilapia rendalli* Boulenger. J Fish Biol 10:575-585.

Clemens WA, Wilby GV. 1961. Fishes of the Pacific coast of Canada. 2nd ed. Bull Fish Res Bd Can 68:443.

Cockell KA, Hilton JW. 1988. Preliminary investigations on the comparative chronic toxicity of four dietary arsenicals to juvenile rainbow trout (*Salmo gairdneri* R.). Aquat Toxicol 12:73-82.

Cockell KA, Bettger WJ. 1993. Investigations of the gallbladder pathology associated with dietary exposure to disodium arsenate heptahydrate in juvenile rainbow trout (*Oncorhynchus mykiss*). Toxicology 77(3):233-248

Cockell KA, Hilton JW, Bettger WJ. 1991. Chronic toxicity of dietary disodium arsenate heptahydrate to juvenile rainbow trout (*Oncorhynchus mykiss*). Arch Environ Contam Toxicol 21:518-527.

Cockell KA, Hilton JW, Bettger WJ. 1992. Hepatobiliary and hematological effects of dietary disodium arsenate heptahydrate in juvenile rainbow trout (*Oncorhynchus mykiss*). Comp Biochem Physiol 103C(3):453-458

Coenen TMM, Brouwer A, Enninga IC, Koeman JH. 1992. Subchronic toxicity and reproduction effects of tri-*n*-butyltin oxide in Japanese quail. Arch Environ Contam Toxicol 23:457-463.

Cohen DM, Inada T, Iwamoto T, Scialabba N. 1990. FAO species catalogue. Vol. 10. Gadiform fishes of the world (Order Gadiformes). An annotated and illustrated catalogue of cods, hakes, grenadiers and other gadiform fishes known to date. FAO Fish. Synop. 125, Vol. 10.

Collier TK, Varanasi U. 1991. Hepatic activities of xenobiotic metabolizing enzymes and biliary levels of xenobiotics in English sole (*Parophrys vetulus*) exposed to environmental contaminants. Arch Environ Contam Toxicol 20:462-473.

Collier TK, Singh SV, YC Awasthi, Varanasi U, 1992. Hepatic xenobiotic metabolizing enzymes in two species of benthic fish showing different prevalences of contaminant-associated liver lesions. Toxicol Appl Pharmacol 113:319-324.

Collier TK, Johnson LL, Myers MS, Stehr CM, Krahn MM, Stein JE. 1998. Fish injury in the Hylebos Waterway of Commencement Bay, Washington. NOAA Tech Memo NMFS-NWFSC-36. Northwest Fisheries Science Center, National Marine Fisheries Service, National Oceanic and Atmospheric Administration, Seattle, WA.

Columbia Basin Fish and Wildlife Authority. 1996. Contamination ecology of selected fish and wildlife of the lower Columbia River. A report to the Bi-State Water Quality Program. Columbia Basin Fish and Wildlife Authority.

Compagno LJV. 1984. FAO species catalogue. Vol. 4. Sharks of the world. An annotated and illustrated catalogue of shark species known to date. Part 1. Hexanchiformes to Lamniformes. FAO Fish. Synop. (125, Vol. 4, Part 1). Food and Agriculture Organization of the United Nations, Rome, Italy.

Cook RH, Pierce RC, Eaton PB, Lao RC, Onuska FI, Payne JF, Vavasour E. 1983. Polycyclic aromatic hydrocarbons in the aquatic environment: formation sources, fate and effects on aquatic biota. NRCC 18981. National Research Council of Canada, Ottowa, ON.

Cordell JR. 2001a. Personal communication (e-mail to Matt Luxon, Windward Environmental LLC regarding LDW ecology). Wetlands researcher, Department of Fisheries, University of Washington, Seattle WA. May 7.

Cordell JR. 2001b. Personal communication (e-mail to Matt Luxon, Windward Environmental LLC regarding observations of juvenile chinook and other wildlife in the LDW). Researcher, Department of Fisheries, University of Washington, Seattle, WA. July 9.

Cordell JR, Tear LM, Jensen K,Simenstad CA, Hood WG. 1994. Duwamish River coastal America restoration and reference sites: results and recommendations from year one pilot and monitoring studies. FRI-UW-9416. Fisheries Research Institute, University of Washington, Seattle, WA.

Cordell JR, Tear LM, Simenstad CA, Hood WG. 1996. Duwamish river coastal America restoration and reference sites: Results from 1995 monitoring studies. FRI-UW-9612. Fisheries Research Institute, University of Washington, Seattle, WA.

Cordell JR, Tear LM, Jensen K, Luiting V. 1997. Duwamish River coastal America restoration and reference sites. Results from 1996 monitoring studies. FRI-UW-9709. Fisheries Research Institute, University of Washington, Seattle WA.

Cordell JR, Tear LM, Jensen K, Higgins HH. 1999. Duwamish River coastal America restoration and reference sites: Results from 1997 monitoring studies. FRI-UW-9903. Fisheries Research Institute, University of Washington, Seattle, WA.

Cordell JR, Tear LM, Jensen K. 2001. Biological monitoring at Duwamish River coastal America restoration and reference sites: A seven-year retrospective. SAFS-UW-0108. Wetlands Ecosystem Team, School of Aquatic and Fisheries Sciences, University of Washington, Seattle, WA.

Coulter W, Adams CM, Decker DJ. 1984. River otter biology. In: New York's Wildlife Resources. Department of Natural Resources, New York State College of Agriculture and Life Sciences, State University at Cornell University, Ithaca, NY. http://www.nyotter.org/pages/biology.html. June 6, 2000.

Courtney CH, Reed JK. 1971. Accumulation of DDT from food and from water by golden shiner minnows, *Notemigonus crysoleucas*. In: 25th Annual Conference, Southeastern Association of Game and Fish Commissioners, 17-20 Oct 1971, Charleston, SC. Southeastern Association of Game and Fish Commissioners, Frankfort, KY. pp 426-431.

Cox G, Francis M. 1997. Sharks and rays of New Zealand. Canterbury University Press, Christchurch, New Zealand.

Cropp T. 1985. Personal communication (as cited in Grette and Salo [1986]). Washington Department of Game, Olympia, WA

Cubbage J, Batts D, Briedenbach S. 1997. Creation and analysis of freshwater sediment quality values in Washington state. Publication 97-323. Technical report. Washington Department of Ecology, Olympia, WA.

Cuerrier JP, Keith JA, Stone E. 1967. Problems with DDT in fish culture operations. Natur Can 94:315-320.

Culhane T, Kelly A, Liszak J. 1995. Initial watershed assessment water resources inventory Area 9 Green-Duwamish Watershed. Northwest Regional Office, Washington Department of Ecology, Bellevue, WA.

Curl HC, Baker ET, Bates TS, Cannon GA, Feely RA, Geiselman TL, Murphy PP, Pashinski DJ, Paulson AJ. 1987. Contaminant transport from Elliott and Commencement Bays. No. 903. Pacific Marine Environmental Laboratory, National Oceanic and Atmospheric Administration, Seattle, WA.

Custer TW, Heinz GH. 1980. Reproductive success and nest attentiveness of mallard ducks fed Aroclor® 1254. Environ Pollut 21:313–318.

Dahlgren RB, Linder RL, Carlson CW. 1972. Polychlorinated biphenyls: their effects on penned pheasants. Environ Health Perspect 1:89-101.

Davies IM. 1999. Personal communication (facsimile to K. Keeley, US Environmental Protection Agency Region 10, Seattle, WA, regarding list of known imposex-affected prosobranch species). Marine Laboratory, Aberdeen, Scotland. April 8.

Davis HG. 1993. Effects of feeding carp from Saginaw Bay, Michigan to river otter. Master's thesis. Michigan State University, East Lansing, MI. 108 pp.

Davis RD, Beckett PHT, Wollan E. 1978. Critical levels of twenty potentially toxic elements in your spring barley. Plant and Soil 49:395-408.

Dawson CE. 1985. Indo-Pacific pipefishes (Red Sea to the Americas). The Gulf Coast Research Laboratory, Ocean Springs, MS.

Day DE. 1976. Homing behavior and population stratifcation in Central Puget Sound English Sole (*Parophrys vetulus*). J Fish Res Board Can 33:287-282.

De Vries TH. 1989. Effecten van PCBs op de voortplanting van marterachtigen. Institute for Environmental Studies, Free University, Amsterdam, The Netherlands.

Delzell E, Giesy J, Munro I, Doull J, Mackay D, Williams G. 1994. Interpretive review of the potential adverse effects of chlorinated organic chemicals on human health and the environment. Regulat Toxicol Pharmacol 20(1):S1-S1056.

DesGranges JL, Rodrigue J, Tardif B, Laperle M. 1998. Mercury accumulation and biomagnification in ospreys (*Pandion haliaetus*) in the James Bay and Hudson Bay regions of Quebec. Arch Environ Contam Toxicol 35:330-341.

Dethier MN, Schoch GC. 2000. The shoreline biota of Puget Sound: extending spatial and temporal comparisons. Prepared for Washington Department of Natural Resources Nearshore Habitat Program.

Dexter RN, Anderson DE, Quinlan E, Goldstein L, Strickland R, Pavlou S, Clayton J, Kocan R, Landolt M. 1981. A summary of knowledge of Puget Sound related to chemical contaminants. National Oceanic and Atmospheric Administration, Boulder, CO.

Di Toro DM, McGrath JA, Hansen DJ. 2000. Technical basis for narcotic chemicals and polycyclic aromatic hydrocarbon criteria. i. water and tissue. Environ Toxicol Chem 19:1951–1970.

Dixon DR, Prosser H. 1986. An investigation of the genotoxic effects of an organotin antifouling compound (bis(tributyltin) oxide) on the chromosomes of the edible mussel, *Mytilus edulis*. Aquat Toxicol 8:185-195.

Dixon RK. 1988. Response of ectomycorrhizal *Quercus rubra* to sol cadmium, nickel and lead. Soil Boil Biochem 20:555-559.

Duke TW, Lowe JL, Wilson AJ Jr. 1970. A polychlorinated biphenyl (Aroclor 1254) in the water, sediment, and biota of Escambia Bay, Florida. Bull Environ Contam Toxicol 5:171-180.

Dunning JB Jr. 1993. CRC handbook of avian body masses. CRC Press, Inc., Boca Raton, FL. 371 pp.

Easton MDL, Luszniak K, Von der Geest E. 2002. Preliminary examination of contaminant loadings in farmed salmon, wild salmon and commercial salmin feed. Chemoshpere 46:1053-1074.

Ecology. 1994. Natural background soil metals concentrations in Washington state. Publication #94-115. Toxics Cleanup Program, Department of Ecology, Olympia, WA.

Ecology. 2000. WRIA 9 report: Historical and current salmonid populations, life histories, and habitat conditions. Draft report NRC.54. King County Department of Natural Resources, Seattle, WA.

Edens FW, Garlich JD. 1983. Lead-induced egg production decrease in leghorn and Japanese quail hens. Poult Sci 62:1757-1763.

Edens FW, Benton E, Bursian SJ, Morgan GW. 1976. Effect of dietary lead on reproductive performance in Japanese quail, *Coturnix coturnix japonica*. Toxicol Appl Pharmacol 38:307-314.

Efroymson RA, Will ME, Suter II GW, Wooten AC. 1997. Toxicological benchmarks for screening contaminants of potential concern for effects on terrestrial plants. 1997 revision. ES/ER/TM-85/R3. Oak Ridge National Laboratory, US Department of Energy, Oak Ridge, TN.

Eisler R. 1980. Accumulation of zinc by marine biota. In: Nriagu JO, ed, Health effects. Part 2. John Wiley and Sons, NY. pp 259-357.

Eisler R. 1985. PAH hazards to fish, wildlife, and invertebrates: A synoptic review. US Fish and Wildlife Service Biological Report 85(1.11). Patuxent Wildlife Research Center, US Fish and Wildlife Service, Laurel, MD.

Eisler R. 1986a. Chromium hazards to fish, wildlife, and invertebrates: A synoptic review. Fish and Wildlife Service, US Fish and Wildlife Service Biological Report 85(1.6). Patuxent Wildlife Research Center, US Fish and Wildlife Service, Laurel, MD.

Eisler R. 1986b. Polychlorinated biphenyl hazards to fish, wildlife, and invertebrates: a synoptic review. US Fish and Wildlife Service Biological Report 85(1.7). Patuxent Wildlife Research Center, US Fish and Wildlife Service, Laurel, MD.

Eisler R. 1987a. Copper hazards to fish, wildlife and invertebrates: A synoptic review. US Fish and Wildlife Service Biological Report. Patuxent Wildlife Research Center, US Fish and Wildlife Service, Laurel, MD.

Eisler R. 1987b. Mercury hazards to fish, wildlife, and invertebrates: A synoptic review. US Fish and Wildlife Service Biological Report 85(1.10) Patuxent Wildlife Research Center, US Fish and Wildlife Service, Laurel, MD.

Eisler R. 1988a. Lead hazards to fish, wildlife, and invertebrates: a synoptic review. US Fish and Wildlife Service Biological Report 85(1.14). Patuxent Wildlife Research Center, US Fish and Wildlife Service, Laurel, MD.

Eisler R. 1988b. Arsenic hazards to fish, wildlife, and invertebrates: a synoptic review. US Fish and Wildlife Service Biological Report 85(1.12). Patuxent Wildlife Research Center, US Fish and Wildlife Service, Laurel, MD.

Eisler R. 1989. Tin hazards to fish, wildlife, and invertebrates: A synoptic review. US Fish and Wildlife Service. Biological Report 85(1.15). Patuxent Wildlife Research Center, US Fish and Wildlife Service, Laurel, MD.

Eisler R. 1993. Zinc hazards to fish, wildlife, and invertebrates: a synoptic review. US Fish and Wildlife Service Biological Report Number 10. Patuxent Wildlife Research Center, US Fish and Wildlife Service, Laurel, MD.

Environmental Solutions Group (ESG). 1999. Waterway sediment operable unit, Harbor Island Superfund site. Assessing human health risks from the consumption of seafood: Human health risk assessment report. Prepared for Port of Seattle, Todd Shipyards, and Lockheed-Martin for submittal to US Environmental Protection Agency, Region 10, Seattle, WA. Environmental Solutions Group Inc., Seattle, WA.

EPA. 1980. Ambient water quality criteria for polynuclear aromatic hydrocarbons. Report 440/5-80-069. 193 pp. US Environmental Protection Agency, Washington, DC.

EPA. 1989. Risk assessment guidance for Superfund, Volume I -- Human health evaluation manual, Part A Interim Final. EPA/540/1-89/002. Office of Emergency and Remedial Response, US Environmental Protection Agency, Washington, DC.

EPA. 1990. Macroinvertebrate field and laboratory methods for evaluating the biological integrity of surface waters. Report No. 600/4-90/030, US Environmental Protection Agency, Office of Research and Development, Cincinnati, OH.

EPA. 1992. Framework for ecological risk assessment. EPA/630/R-92/001. Risk Assessment Forum, US Environmental Protection Agency, Washington, DC.

EPA. 1993a. Technical basis for deriving sediment quality criteria for nonionic organic contaminants for the protection of benthic organisms by using equilibrium partitioning. EPA 922-R-93-011. Office of Science and Technology, US Environmental Protection Agency, Washington, DC.

EPA. 1993b. Wildlife exposure factors handbook. EPA/600/R-93/187a. Office of Research and Development, US Environmental Protection Agency, Washington DC.

EPA. 1994. Ecological risk assessment guidance for Superfund: Process for designing and conducting ecological risk assessments. Environmental Response Team, US Environmental Protection Agency, Edison, NJ.

EPA. 1995. OPPT chemical fact sheet: Chlorobenzene. US Environmental Protection Agency. http://www.epa.gov/opptintr/chemfact/chlor-sd.txt. May 21, 2001.

EPA. 1996. Calculation and evaluation of sediment effect concentrations for the amphipod *Hyalella azteca* and the midge *Chironomus riparius*. EPA 905-R96-008. US Environmental Protection Agency, Chicago, IL.

EPA. 1997a. Ecological risk assessment guidance for Superfund: Process for designing and conducting ecological risk assessments. EPA/540/R-97/006. Environmental Response Team, US Environmental Protection Agency, Edison, NJ.

EPA. 1997b. EPA Region 10 supplemental ecological risk assessment guidance for Superfund. EPA/910/R-97/005. Risk Evaluation Unit, Office of Environmental Assessment, US Environmental Protection Agency Region 10, Seattle, WA.

EPA 1997c. Mercury study report to congress. Volume III: Fate and transport of mercury in the environment. Appendix D. EPA-452/R-97-005. US Environmental Protection Agency, Office of Air Quality Planning & Standards and Office of Research and Development, Washington DC.

EPA. 1998a. Guidelines for ecological risk assessment. EPA/630/R-95/002-F. Risk Assessment Forum, US Environmental Protection Agency, Washington, DC.

EPA. 1998b. Human health risk assessment protocol, Appendix A-2. Target organs and critical effects for compounds with reference dose values. USEPA Region 6, Dallas, TX.

EPA. 1999. Development of a tissue trigger level for bioaccumulated tributyltin in marine benthic organisms: West Waterway Harbor Island Superfund Site Seattle WA. US Environmental Protection Agency Region 10 Seattle WA.

EPA. 2000a. Evaluation of dermal contact and inhalation exposure pathways for the purpose of setting Eco-SSLs. Draft ecological soil screening level guidance, Exhibit 1-3. US Environmental Protection Agency, Washington, DC.

EPA. 2000b. Hudson River PCBs reassessment. Revised baseline ecological risk assessment. US Environmental Protection Agency Region 2, New York, NY.

EPA. 2000c. Hudson River PCBs reassessment RI/FS. Response to peer review comments on the baseline ecological risk assessment. US Environmental Protection Agency Region 2, New York, NY.

EPA. 2002. Role of background in the CERCLA Cleanup Program. OSWER 9285.6-07P. Office of Solid Waste and Emergency Response, US Environmental Protection Agency, Washington, DC.

Eschmeyer WN, Herald ES, Hammann H. 1983. Pacific coast fishes. Peterson Field Guide Series. Houghton Mifflin, Boston, MA.

Evans ML. 1980. Copper accumulation in the crayfish (*Orconectes rusticus*). Bull Environ Contam Toxicol 24:916-920.

Everitt RD, Gearin PJ, Skidmore JS. 1981. Prey items of harbor seals and California sea lions in Puget Sound, Washington. Murrelet 62:83-8

EVS. 1995. Fish collection field log. Elliott Bay/Duwamish River fish tissue investigation. EVS Environment Consultants, Inc., Seattle, WA.

EVS, NOAA. 1998. Sheboygan River and Harbor aquatic ecological risk assessment. Prepared for US Environmental Protection Agency Region 5. EVS Environment Consultants, Inc. and National Oceanic and Atmospheric Administration, Seattle, WA.

EVS Solutions. 1999. Review of tissue residue effects data for tributyltin, mercury, and polychlorinated biphenyls. Prepared for the Port of Seattle, Lockheed Martin, and Todd Pacific Shipyards for submittal to US Environmental Protection Agency Region 10. EVS Solutions Inc., Seattle, WA.

EXTOXNET. 1996. Pesticide information profiles, Extension Toxicology Network. http://ace.ace.orst.edu/info/extoxnet/. April 30, 2001.

Fabacher DL, Besser JM, Schmitt CJ, Harshbarger JC, Peterman PH, Lebo JA. 1991. Contaminated sediments from tributaries of the Great Lakes: Chemical characterization and carcinogenic effects in medaka (*Oryzias latipes*). Arch Environ Contam Toxicol 20:17-34.

Fairey R, Long ER, Roberts CA, Anderson BS, Phillips BM, Hunt JW, Puckett HM, Wilson CJ. 2001. An evaluation of methods for calculating mean sediment quality guideline quotients as indicators of contamination and acute toxicity to amphipods by chemical mixtures. Environ Toxicol Chem 20(10):276-2286.

Feder HM. 1980. Distribution, abundance, community structure, and trophic relationships of the benthos of the northeastern Gulf of Alaska from Yakutat Bay to Cross Sound. In: Environmental assessment of the Alaskan continental shelf, Annual Reports 1:597-648. Office of Marine Pollution Assessment, National Oceanic and Atmospheric Administration, Juneau, AK.

Feng L, Wang L, Zhao Y, Song B. 1996. Effects of substituted anilines and phenols on root elongation of cabbage seed. Chemosphere 32: 1575-1583.

Ferenczy J, Szegletes T, Balint T, Abraham M, Nemcsok J. 1997. Characterization of acetylcholinesterase and its molecular forms in organs of five freshwater teleosts. Fish Physiol Bichem 16:515-529.

Fernie, KJ, Smite JE, Bortolotti GR, Bird DM. 2001. In ovo exposure to polychlorinated biphenyls: reproductive effects on second-generation American kestrels. Arch Environ Contam Toxicol 40:544-550.

Fiksel J, Cooper C, Eschenroeder A, Goyer M, Perwak J. 1981. Exposure and risk assessment for cyanide. EPA/440/4-85/008. NTIS PB85-220572. US Environmental Protection Agency, Washington, DC.

Finley MT, Dieter MP, Locke LN. 1976. Sublethal effects of chronic lead ingestion in mallard ducks. J Toxicol Environ Health 1:929-937.

Firestone, D. 1973. Etiology of chick edema disease. Environ Health Perspect 5:59-66.

Fischer DL, Hancock GA. 1997. Interspecies extrapolation of acute toxicity in birds: body scaling vs. phylogeny. Poster presented at 1997 SETAC meeting, San Francisco, CA.

Fisher JP, Spitsbergen JM, Bush B, Jahan-Parwar B. 1994. Effect of embryonic PCB exposure on hatching success, survival, growth and developmental behavior in landlocked Atlantic salmon, *Salmo salar*. In: Gorsuch JW, Dwyer FJ, Ingersoll CG, La Point TW, eds, Environmental toxicology and risk assessment. Vol 2. ASTM STP 1216. American Society for Testing and Materials, Philadelphia PA, pp 298-314.

Fitch JE, Lavenberg RJ, 1975. Tidepool and nearshore fishes of California. California Natural History Guides: 38. University of California Press, Berkeley and Los Angeles, CA.

Fitzhugh OG. 1948. Use of DDT insecticides on food products. Ind Eng Chem 40:704-705.

Fitzsimmons T. 1999. Personal communication (letter to Interested Persons describing the halting of the SMS rule amendment process). Director, Washington Department of Ecology, Lacey, WA. December 30.

Fleming WJ, Ailstock MS, Momo JJ. 1995. Net photosynthesis and respiration of Sago pondweed exposed to herbicides. In: Huges JS, Biddinger GR, Mones E. 1995. Environmental Toxicolgy and Risk Assessment: Vol. 3. ASTM STP 1218. American Society for Testing and Materials, Phildelphia, PA.

Forrester CR. 1969. Life history on some ground fish species. Fish Res Bd Can Technical Report 105:1-17.

Fraley JJ, Shepard BB. 1989. Life history, ecology and population status of migratory bull trout (*Salvelinus confluentus)* in the Flathead Lake and River system, Montana. Northwest Sci 63(4):133-143.

Freeman HC, Idler DR. 1975. The effect of polychlorinated biphenyl on steroidogenesis and reproduction in the brook trout (*Salvelinus fontinalis*). Can J Biochem 53:666-670.

Freidig AP, Hermens JLM. 2000. Chapter 7: An elementary pharmacodynamic model (EPD) for the analysis of time dependent aquatic toxicity data of reactive chemicals: Habers Law revisited. In: Freidig AP, ed, Models for risk assessment of reactive chemicals in aquatic toxicology. Tekst. - Proefschrift Universiteit Utrecht.

Frontier. 1996. Mercury results in 18 fish samples for the Elliott Bay/Duwamish River Project. Frontier Geosciences, Seattle, WA.

Fuchsman PC, Barber TR, Sheehan PJ. 1999. Sediment toxicity evaluation for hexachlorobenzene: spiked sediment tests with *Leptocheirus plumulosus, Hyalella azteca,* and *Chironomus tentans*. Environ Sci Technol 33:573-579.

Fuller GB, Hobson WC. 1986. Effect of PCBs on reproduction in mammals. In: Waid JS, ed, PCBs and the environment. Vol II. CRC Press, Inc., Boca Raton, FL.

Gall OE, Barnette RM. 1940. Toxic limits of replaceable zinc to corn and cowpeas grown on three Florida soils. J Amer Soc Agronomy 32:23-32.

Galvez F, Wood CM. 1999. Physiological effects of dietary silver sulfide exposure in rainbow trout. Environ Toxicol Chem 18(1):84-88.

Gasaway WC, Buss IO. 1972. Zinc toxicity in the mallard duck. J Wildl Manag 36:1107-1117.

Gibbs PE, Pascoe PL, Burt GR. 1988. Sex change in the female dog whelk, *Nucella lapillus,* induced by tributyltin from antifouling paints. J Mar Biol Assoc UK 68:715-731.

Gibbs PE, Bryan GW, Pascoe PL, Burt GR. 1990. Reproductive abnormalities in female *Oceneba erinacea* (gastropoda) resulting from tributyltin-induced imposex. J Mar Biol Assoc UK 70:639-656.

Giesy JP, Ludwig JP, Tillitt DE. 1994a. Deformities in birds of the Great Lakes region - assigning causality. Environ Sci Technol 28(3).

Giesy JP, Ludwig JP, Tillitt DE. 1994b. Dioxins, dibenzofurans, PCBs and colonial, fish-eating water birds. In: Shecter A, ed, Dioxins and health, 249:249.

Gilani SH, Morano M. 1979. Chromium poisoning and chick embryogenesis. Environ Res 19:427-431.

Gilbert FF, Nancekivell EG. 1982. Food habits of mink (*Mustela vison*) and otter (*Lutra canadensis*) in northeastern Alberta. Can J Zool 60:1282-1288.

Goettl JP Jr, Davies PH. 1976. Water pollution studies. Job progress report, Federal Aid Project F-33-R-11. Department of Natural Resources, Boulder, CO.

Goettl JP, Davies PH, Sinley JR. 1976. Water pollution studies. Colorado Fisheries Research Review 1972-1975. CO Div Wildlife Rev 8:68-75.

Goetz F. 1989. Biology of the bull trout *Salvelinus confluentus*, a literature review. Willamette National Forest, Eugene, Oregon. (As referenced in USDI, FWS 1997).

Golub MS, Donald JM, Reyes JA. 1991. Reproductive toxicity of commercial PCB mixtures: LOAELs and NOAELs from animal studies. Environ Health Perspect 94:245-253.

Gordon CD. 1965. Aspects of the life-history of *Cymatogaster aggregata* Gibbons. MS thesis, University of British Columbia, Vancouver, BC.

Greer KR. 1955. Yearly food habits of the river otter in the Thompson Lakes region, northwestern Montana, as indicated by scat analyses. Am Midl Nat 54:299-313.

Grette GB, Salo EO. 1986. The status of anadromous fishes of the Green/Duwamish River system. Final Report. Prepared for the US Army Corps of Engineers, Seattle District. Evans Hamilton Inc., Seattle, WA.

Gries TH. 1997. Revisions to DMMP screening and maximum level guidelines: an issue paper prepared for the CSMP agencies. Sediment Management Unit, Washington Department of Ecology, Lacey, WA.

Gries TH. 1999. Personal communication (handouts for June 16, 1999 SMS Implementation Committee Meeting, Lacey, WA). Sediment Management Unit, Washington Department of Ecology, Lacey, WA.

Gries TH, Waldow KH. 1996. Progress re-evaluating Puget Sound apparent effects thresholds (AETs). Vol I: 1994 amphipod and echinoderm larval AETs. Prepared for Puget Sound Dredged Disposal Analysis (PSDDA) agencies by Washington Department of Ecology, Olympia, WA.

Groot C, Margolis L. 1998. Pacific salmon life histories. UBC Press, Vancouver, BC.

Grossman, G. D. 1979. Demographic characteristics of an intertidal bay goby (*Lepidogobius lepidus*). Environ Biol Fish 4(3):207-218.

Gunther P, Pestermer W. 1990. Risk assessment for certain xenobiotics by bioassay method with higher plants. Environ Manag. 14:381-388.

Gutleb AC, Kranz A. 1998. Estimation of polychlorinated biphenyl (PCB) levels in livers of the otter (*Lutra lutra*) from concentrations in scats and fish. Water Air Soil Pollut 106:481-491.

Hagemeyer J, Lohrmann D, Breckle SW. 1993. Development of annual xylem rings and shoot growth of young beech (*Fagus sylvatica* L.) grown in soil with various Cd and Zn levels. Water, Air, and Soil Pollut 69:351-361.

Hamelink JL, Waybrant RC, Ball RC. 1971. A proposal: Exchange equilibria control the degree chlorinated hydrocarbons are biologically magnified in lentic environments. Trans Am Fish Soc 100:207-214.

Handy RD. 1992. The assessment of episodic metal pollution. 1. Uses and limitations of tissue contaminant analysis in rainbow trout (*Oncorhynchus mykiss*) after short waterborne exposure to cadmium or copper. Arch Environ Contam Toxicol 22:74-81.

Handy RD. 1993. The accumulation of dietary aluminium by rainbow trout, *Oncorhynchus mykiss*, at high exposure concentrations. J Fish Biol 42:603-606.

Hansen DJ, Parrish PR, Lowe JL, Wilson Jr AJ, Wilson PD. 1971. Chronic toxicity, uptake, and retention of Aroclor 1254 in two estuarine fishes. Bull Environ Contam Toxicol 6:113-119.

Hansen D, Schimmel SC, Forester J. 1973. Aroclor 1254 in eggs of sheepshead minnows: effect on fertilization success and survival of embryos and fry. In: Proceedings of 27th Annual Conference, Southeastern Association of Game and Fish Commissioners, Hot Springs, AR, October 14-17, 1973, pp. 420-426.

Hansen DJ, Schimmel SC, Forester J. 1974. Aroclor 1016: Toxicity to and uptake by estuarine animals. Environ Res 7:363-373.

Hansen DJ, Schimmel SC, Forester J. 1975. Effects of Aroclor 1016 on embryos, fry, juveniles, and adults of sheepshead minnows (*Cyprinodon variegates*). Trans Am Fish Soc 104:584-588.

Harper-Owes. 1981. Duwamish Estuary study. Task II report: Pollutant loads to the Duwamish Estuary, 1970-1980. Prepared for Municipality of Metropolitan Seattle, Water Quality Division. Harper-Owes Company, Seattle, WA.

Harper-Owes. 1983. Water quality assessment of the Duwamish Estuary, Washington. Prepared for Municipality of Metropolitan Seattle, Water Quality Division. Harper-Owes Company, Seattle, WA.

Hart Crowser. 1998. Dredge material characterization, Hurlen Construction Company and Boyer Alaska Barge Lines berthing area. Hart Crowser, Inc., Seattle, WA.

Hart DR, Heddle JA. 1991. Micronucleus assays in peripheral blood of rainbow trout: Timing of response and chemical mutagen sensitivity. In: Chapman P, Bishay F, Power E, Hall K, Harding L, McLeay D, Nassichuk M, Knapp K, eds, Proceedings of the 17th Annual Aquatic Toxicity Workshop, Nov.5-7, 1990, Vancouver, BC. Can Tech Rep Fish Aquat Sci 1774(2):993-1010.

Hart JL. 1973. Pacific fishes of Canada. Bull. 180. Fisheries Research Board of Canada, Ottawa, ON.

Hartman FA. 1961. Locomotor mechanisms in birds. Smithsonian Misc Coll 143, Washington, DC.

Hartman. 1992. Lonestar Northwest Terminal, Duwamish River PSDDA sampling and analysis results. Prepared for Lone Star Northwest. Hartman Associates Inc., Seattle, WA.

Hartman. 1995. Kaiser dock upgrade Duwamish Waterway PSDDA sampling and analysis results. Prepared for Lone Star Northwest and James Hardie Gypsum. Hartman Associates Inc., Seattle, WA.

Haseltine SD, Prouty RM. 1980. Aroclor 1242 and reproductive success of adult mallards (*Anas platyrhynchos*). Environ Res 23:29-34.

Hassett JJ, Miller JE, Keoppe DE. 1976. Interaction of lead and cadmium on maize root growth and uptake of lead and cadmium by roots. Environ Pollut 11:297-302.

Hatakeyama S, Yasuno M. 1987. Chronic effects of Cd on the reproduction of the guppy (*Poecilia reticulata*) through Cd-accumulated midge larvae (*Chironomus yoshimatsui*). Ecotoxicol Environ Safety 14:191-207.

Hattula ML, Karlog O. 1972. Toxicity of PCBs to goldfish. Acta Pharmacol Toxicol 31:238-240.

Hawryshyn, CW, Mackay, WC, and Nilsson TH. 1982. Methyl mercury induced visual deficits in rainbow trout. Canad J Zool 60:3127-3133.

Healy MC. 1991. Life history of chinook salmon (*Oncorhynchus tshawytscha*). In: Groot C, Margolis L, eds, Pacific salmon life histories. UBC Press, Vancouver, BC, pp 311-394.

Heath RG, Spann JW, Kreitzer JF, Vance C. 1972. Effects of polychlorinated biphenyls on birds. In: Voous KH, ed, Proceedings of the XV International Ornithological Congress, August 30–September 5, 1970. Brill EJ, Leiden. pp. 475–485.

Heinz GH. 1979. Methylmercury: reproductive and behavioral effects on three generations of mallard ducks. J Wildl Manage 43(2).

Heinz GH. 1980. Eggshell thickness in mallards fed methylmercury. Bull Environ Contam Toxicol 25:498-502.

Heinz GH, Hoffman DJ, Gold LG. 1989. Impaired reproduction of mallards fed an organic form of selenium. J Wildl Manag 53:418-428.

Heisinger JF, Green W. 1975. Mercuric chloride uptake by eggs of the ricefish and resulting teratogenic effects. Bull Environ Contam Toxicol 14:665-673.

Heisinger JF, Hansen CD, Kim JH. 1979. effect of selenium dioxide on the accumulation and acute toxicity of mercuric chloride in goldfish. Arch Environ Contam Toxicol 8:279-283.

Hendricks JD, Scott WT, Putnam TP, Sinnhuber RO. 1981. Enhancement of aflatoxin B1 hepatocarcinogenesis in rainbow trout (*Salmo gairdneri*) embryos by prior exposure of gravid females to dietary Aroclor 1254. In: Branson DR, Dickson KL, eds, Aquatic Toxicology and Hazard Assessment. Fourth Conference. ASTM STP 737. American Society of Testing and Materials, Philadelphia, PA, pp 203-214.

Hendricks JD, Meyers TR, Shelton DW, Casteel JL, Bailey GS. 1985. Hepatocarcinogenicity of benzo[a]pyrene to rainbow trout by dietary exposure and intraperitoneal injection. J Natl Cancer Inst 74:839-851.

Henny CJ, Blus LJ, Gregory SV, Stafford CJ. 1981. PCBs and organochlorine pesticides in wild mink and river otters from Oregon. In: Chapman JA, Pursley D, eds, Proceedings of the Worldwide Furbearer Conference, Vol III, August 3-11, 1980. Center for Environmental and Estuarine Studies, University of Maryland, Frostburg, MD, pp. 1763-1779.

Hoffman DJ, Gay ML. 1981. Embryotoxic effects of benzo(a)pyrene, chrysene, and 7,12-dimethylbenz(a)anthracene in petroleum hydrocarbon mixtures in mallard ducks. J Toxicol Environ Health 7:775-787.

Hoffman DJ, Franson JC, Pattee OH, Bunck CM, Anderson A. 1985. Survival, growth, and accumulation of ingested lead in nestling American kestrels (*Falco sparverius*). Arch Environ Contam Toxicol 14:89-94.

Hoffman DJ, Rattner BA, Burton Jr GA, Cairns Jr J. 1995. Handbook of ecotoxicology. Lewis Publishers, Boca Raton, FL, pp. 367-377.

Hoffman DJ, Rice CP, Kubiak TJ. 1996. PCBs and dioxins in birds. In: Beyer WN, Heinz GH, Redmon-Norwood AW, eds, Environmental contaminants in wildlife. Lewis Publishers, Boca Raton, FL, pp 165–207.

Hoffman DJ, Melancon MJ, Klein PN, Eisemann JD, Spann JW. 1998. Comparative developmental toxicity of planar polychlorinated biphenyl congeners in chickens, American kestrels, and common terns. Environ Toxicol Chem 17(4):747-757.

Hoffman E. 2001. Personal communication (e-mail to Kathy Godtfredsen regarding crab residue-effects data). Sediment Management Program, US Environmental Protection Agency Region 10, Seattle, WA. December 20.

Hoffman RD. 1978. The diets of herons and egrets in southwestern Lake Erie. In: Sprunt A, Oge J, Winckler S, eds, Wading birds. Research report No. 7, National Audubon Society, New York, NY, pp 365-369.

Holland GA. 1954. A preliminary study of the populations of English sole in Carr Inlet and other locations in Puget Sound. MS Thesis. University of Washington, Seattle, WA.

Hoover A. 1988. Harbor seal, *Phoca vitulina*. In: Lentfor JW, ed, Selected marine mammals of Alaska: Species accounts with research and management recommendations. Marine Mammal Commission, Washington, DC, pp 125-157.

Hopkins CL, Solly SRB, Ritchie AR. 1969. DDT in trout and its possible effect on reproductive potential. NZ J Mar Freshwater Res 3:220-229.

Hornshaw, TC, Aulerich RJ, Johnson HE. 1983. Feeding Great Lakes fish to mink: effects on mink and accumulation and elimination of PCBs by mink. J Toxicol Environ Health 11:933-946.

Huang X-D, Dixon DG, Greenberg BM. 1991. Photoinduced toxicity of polyciclic aromatic hydrocarbons to the higher plant *Lemna gibba* L. G-3. In: Gorsuch JW, Lower WR, Lewis MA, Wang W, eds, Plants for toxicity assessment. Vol 2. ASTM STP 1115. American Society for Testing and Materials, Philadelphia, PA, pp 209-216.

Hyland JL, Van Dolah RF, Snoots TR. 1999. Predicting stress in benthic communities of southeastern US estuaries in relation to chemical contamination of sediments. Environ Toxicol Chem 18(11):2557-2564.

Ingersoll CG, Haverland PS, Brunson EL, Canfield TJ, Dwyer FJ, Henke CE, Kemble NE, Mount DR, Fox RG. 1996. Calculation and evaluation of sediment effect concentrations for the amphipod *Hyalella azteca* and the midge *Chironomus riparius*. J Great Lakes Res 22(3):602-623.

Ishida M, Suyama K, Adachi S, Hoshino T. 1982. Distribution of orally administered di-ethylhexyl phthalate in laying hens. Poult Sci 61:262-267.

Iversen JA. 1972. Basal energy metabolism of mustelids. J Comp Physiol 81:341-344 (as cited in EPA [1993b]).

James VA, Wigham T. 1986. The effects of cadmium on prolactin cell activity and plasma cortisol levels in the rainbow trout (*Salmo gairdneri*). Aquat Toxicol 8:273-280.

Jana S, Choudhuri MA 1982. Senescence in submerged aquatic angiosperms: Effects of heavy metals. New Phytol 90:477-484. In Losch R, Kohl KI. 1999. Plant respiration under the influence of heavy metals. In Prasad MNV, Hagemeyer J, eds, Heavy metal stress in plants, from molecules to ecosystems. Springer-Verlag, Berlin Heidelberg, pp 139-156.

Janz DM, Bellward GD. 1996. In ovo 2,3,7,8-tetrachlorodibenzo-p-dioxin exposure in three avian species 1. Effects on thyroid hormones and growth during the perinatal period. Toxicol Appl Pharm 139:281-291.

Jarvinen AW, Ankley GT. 1999. Linkage of effects to tissue residues: Development of a comprehensive database for aquatic organisms exposed to inorganic and organic chemicals. SEATAC Press, Pensacola, FL.

Jarvinen AW, Hoffman MJ, Thorslund TW. 1976. Toxicity of DDT food and water exposure to fathead minnows. Duluth MN: US Environmental Protection Agency. EPA-600/3-76/114.

Jarvinen AW, Hoffman MJ, Thorslund TW. 1977. Long-term toxic effects of DDT food and water exposure on fathead minnows (*Pimephales promalas*). J Fish Res Board Can 34:2089-2103.

Jeanes ED, Hilgert PJ. 2000. Juvenile salmonid use of lateral stream habitats in the Middle Green River, Washington. Report prepared for US Army Corps of Engineers, Seattle District and City of Tacoma Public Utilities. R2 Resource Consultants, Redmond, WA.

Jeffries S. 2001. Personal communication (telephone conversation with Berit Bergquist, Windward Environmental LLC, Seattle, WA). Research Scientist, Washington Department of Fish and Wildlife, Olympia, WA. October 29.

Jennings JR, Rainbow PS. 1979. Studies on the uptake of cadmium by the crab *Carcinus maenas* in the laboratory. I. Accumulation from seawater and a food source. Marine Biology 50:131-139.

Jensen S, Kihlstrom JE, Olsson M, Lundberg C, Orberg J. 1977. Effects of PCB and DDT on mink (*Mustela vison*) during the reproductive season. Ambio 6(4):239.

John MK, van Laerhoven C. 1972. Lead uptake by lettuce and oats as affected by lime, nitrogen and sources of lead. J Environ Quality 1:169-171.

Johnson HE, Pecor C. 1969. Coho salmon mortality and DDT in Lake Michigan. Thirty-fourth North American Wildlife Conference: proceedings, Washington, DC, March 2-5, 1969, pp 159-166.

Johnson LL. 2000. An analysis in support of sediment quality thresholds for polycyclic aromatic hydrocarbons (PAHs) to protect estuarine fish. Memorandum to Rachel Friedman, Steven Landino, dated July 24, 2000. Environmental Conservation Division, Northwest Fisheries Science Center, National Marine Fisheries Service, National Oceanic and Atmospheric Administration, Seattle, WA.

Johnson LL, Landahl JT. 1994. Chemical contaminants, liver disease, and mortality rates in English sole (*Pleuronectes vetulus*). Ecolog Applic 4:59-68.

Johnson LL, Casillas E, Collier TK, McCain BB, Varanasi U. 1988. Contaminant effects on ovarian development in English sole (*Pleuronectes vetulus*) from Puget Sound, Washington. Can J Fish Aquat Sci 45:2133-2146.

Johnson LL, Casillas E, Sol SY, Collier TK, Stein JE, Varanasi U. 1993. Contaminant effects on reproductive success in selected benthic fish species. Mar Env Res 35:165-170.

Johnson LL, Sol SY, Lomax DP, Nelson GM, Sloan CA, Casillas E. 1997. Fecundity and egg weight in English sole, *Pleuronectes vetulus*, from Puget Sound, Washington: Influence of nutritional status and chemical contaminants. Fish Bull 95:231-249.

Johnson LL, Landahl JT, Kubin LA, Horness BH, Myers MS, Collier TK, Stein JE. 1998. Assessing the effects of anthropogenic stressors on Puget Sound flatfish populations. J Sea Res 39:125-137.

Johnson LL, Sol SY, Ylitalo GM, Hom T, French B, Olson OP, Collier TK. 1999. Reproductive injury in English sole (*Pleuronectes vetulus*) from the Hylebos Waterway, Commencement Bay, Washington. J Aquat Ecosystem Stress and Recovery 6:289-310.

Johnson LL, Collier TK, Stein JE. 2002. An analysis in support of sediment quality thresholds for polycyclic aromatic hydrocarbons (PAHs) to protect estuarine fish. Aquat Conserv: Marine Freshw Ecosyst 12(5):517-538.

Jones PD, Giesy JP, Newsted JL, Verbrugge DA, Beaver DL, Ankley GT, Tillitt DE, Lodge KB, Niemi GJ. 1993. 2,3,7,8-tetrachlorodibenzo-*p*-dioxin equivalents in tissues of birds at Green Bay Wisconsin, USA. Arch Environ Contam Toxicol 24:345-354.

Jones PD, Giesy JP, Newsted JL, Verbrugge DA, Ludwig JP, Ludwig ME, Auman HJ, Crawford R, Tillitt DE, Kubiak TJ, Best DA. 1994. Accumulation of 2,3,7,8-tetrachlorodibenzo-*p*-dioxin equivalents in double-crested cormorant chicks in the North American Great Lakes. Ecotoxicol Environ Safety 27:192-209.

Kabata-Pendias A, Pendias H. 1984. Trace elements in soils and plants. CRC Press, Inc. Boca Raton, FL.

Kaczynski V, Palmisano J. 1992. A review of management and environmental factors responsible for the decline and lack of recovery of Oregon's wild anadromous salmonids. Report to Oregon Forest Industries Council, Salem, OR.

Kamunde CN, Grosell M, Lott JNA, Wood CM. 2001. Copper metabolism and gut morphology in rainbow trout (*Oncorhynchus mykiss*) during chronic sublethal dietary copper exposure. Can J Fish Aquat Sci 58:293-305.

Kendall RJ, Scanlon PF. 1982. The toxicology of ingested lead acetate in ringed turtle doves *Streptopelia risoria*. Environ Pollut (Series A) 27:255-262.

Kennedy SW, Lorenzen A, Jones SP, Hahn ME, Stegeman JJ. 1996. Cytochrome P4501A induction in avian hepatocyte cultures: a promising approach for predicting the sensitivity of avian species to toxic effects of halogenated aromatic hydrocarbons. Toxicol Appl Pharm 141:214-230

Kenney MJ. 1982. Final fish and wildlife coordination act report on the East, West and Duwamish Waterways, navigation improvement study. Division of Ecological Services, US Fish and Wildlife Service, Olympia, WA.

Kerwin J, Nelson TS (eds). 2000. Habitat limiting factors and reconnaissance assessment report, Green/Duwamish and Central Puget Sound watersheds (WRIA 9 and Vashon Island). Washington Conservation Commission, Olympia, WA, and King County Department of Natural Resources, Seattle, WA.

Khan DH, Frankland B. 1983. Effects of cadmium and lead on radish plants with particular reference to movement of metals through soil profile and plant. Plant and Soil 70:335-345.

Khan DH, Frankland B. 1984. Cellulolytic activity and root biomass production in some metal-contaminated soils. Environ Pollut (Series A) 33:63-74.

Khera, KS. 1979. Teratogenic and genetic effects of mercury toxicity. In: Nriagu JO, ed, The biogeochemistry of mercury in the environment. Elsevier/North-Holland Biomedical Press, New York, NY, pp 501-518.

Kihlstrom JE, Olsson M, Jensen S, Johansson A, Ahlbom J, Bergman A. 1992. Effects of PCB and different fractions of PCB on the reproduction of the mink (*Mustela vison*). Ambio 21(8):563-569.

Kimbrough RD. 1985. Laboratory and human studies on polychlorinated biphenyls (PCBs) and related compounds. Environ Health Perspect 59:99-106.

Kimbrough RD. 1987. Human health effects of polychlorinated biphenyls (PCBs) and polybrominated biphenyls (PBBs). Ann Rev Pharmacol Toxicol 27:87-111.

Kimmel CA, Grant LD, Sloan CS. 1980. Chronic low-level lead toxicity in the rat. 1. Maternal toxicity and perinatal effects. Toxicol Appl Phamacol 56:28-41

King County. 1995. Water quality assessment. King County Department of Natural Resources, Seattle, WA.

King County. 1999a. King County combined sewer overflow water quality assessment for the Duwamish River and Elliott Bay. Volume 1: Overview and interpretation. King County Department of Natural Resources, Seattle, WA.

King County. 1999b. King County combined sewer overflow water quality assessment for the Duwamish River and Elliott Bay. Appendix A: Problem formulation, analysis plan, and field sampling work plan. King County Department of Natural Resources, Seattle, WA.

King County. 1999c. King County combined sewer overflow water quality assessment for the Duwamish River and Elliott Bay. Appendix B2 B3 & B4: Human health wildlife and aquatic life risk assessments. King County Department of Natural Resources, Seattle, WA.

King County. 1999d. King County combined sewer overflow water quality assessment for the Duwamish River and Elliott Bay. Appendix C: Issue Papers. King County Department of Natural Resources, Seattle, WA.

King County. 2000. Duwamish/Diagonal CSO/SD site assessment report. Draft. Elliott Bay/Duwamish Restoration Program. King County Department of Natural Resources, Seattle, WA.

King County, Washington Conservation Commission, and WRIA 9 Steering Committee. 2000. The habitat limiting factors and reconnaissance report, in the Green/Duwamish and Central Puget Sound watersheds (WRIA 9 and Vashon Island). Part V, Appendix. Historical and current salmonid populations. Final Version. King County Department of Natural Resources, Washington Conservation Commission, and WRIA 9 Steering Committee, Seattle, WA.

Kirkpatrick CM. 1940. Some foods of young great blue herons. Am Midl Nat 24: 594-601.

Kishino T, Kobayashi K. 1995. Relation between toxicity and accumulation of chlorophenols at various pH, and their absorption mechanism in fish. Water Res 29: 431-442.

Knight RL, Randolph PJ, Allen GT, Young LS, Wigen RJ. 1990. Diets of nesting bald eagles, *Haliaeetus leucocephalus*, in western Washington. Can Field-Nat 104(4):545-551.

Kobayashi K, Akitake K, Manabe K. 1979. Relation between toxicity and accumulation in goldfish. Bull Jpn Soc Sci Fish 45: 173-175.

Krausmann JD. 2002a. Personal communication (telephone conversation with Berit Bergquist, Windward Environmental, regarding great blue heron use of the LDW). Fish and Wildlife Biologist, US Fish and Wildlife Service, Lacey, WA. March 26.

Krausmann JD. 2002b. Preliminary exposure assessment of dioxin-like chlorobiphenyls in great blue herons of the lower Duwamish River in Seattle, Washington. US Fish and Wildlife Service, Lacey, WA.

Kubin LA. 1997. Growth of juvenile English sole exposed to sediments amended with aromatic compounds. Master's thesis. Western Washington State University, Bellingham, WA. 99 pp.

Kudo A, Mortimer DC. 1979. Pathways for mercury uptake by fish from bed sediments. Environ Pollut 19:239-245.

Kurta A. 1995. Mammals of the Great Lakes region. University of Michigan Press, Ann Arbor, MI.

Kushlan JA. 1978. Feeding ecology of wading birds. In: Sprunt A, Ogden JC, Winckler S, eds, Wading birds. National Audubon Society, New York, NY, pp 249-297.

Lamb A, Edgell P. 1986. Coastal fishes of the Pacific northwest. Harbour Publishing Co. Ltd., Madeira Park, BC.

Landrum PF, Eadie BJ, Faust WR. 1991. Toxicokinetics and toxicity of a mixture of sediment-associated polycyclic aromatic hydrocarbons to the amphipod *Diporeia* sp. Env Tox Chem 10:35-46.

Lanno RP, Slinger SJ, Hilton JW. 1985a. Maximum tolerable and toxicity levels of dietary copper in rainbow trout (*Salmo gairdneri* Richardson). Aquaculture 49:257-268.

Lanno RP, Slinger SJ, Hilton JW. 1985b. Effect of ascorbic acid on dietary copper toxicity in rainbow trout (*Salmo gairdneri* Richardson). Aquaculture 49:269-287.

Larsen DN. 1984. Feeding habits of river otters in coastal southeastern Alaska. J Wildl Manag 484:14460-1452.

Lata K, Veer B. 1990. Phytotoxicity of Zn amended soil to *Spinacia* and *Coriandrum*. Acta Botanica Indica 18:194-198.

Leland HV, Kuwabara JS. 1985. Trace metals. In: Rand GM, Petrocelli SR, eds, Fundamentals of aquatic toxicology: Methods and applications. Hemisphere Publishing Corporation, New York, NY.

Leon H. 1980. Terminal 107 environmental studies. Benthic community impact study for Terminal 107 (Kellogg Island) and vicinity. Prepared for Port of Seattle. Pacific Rim Planners Inc., Seattle, WA.

Lieb AJ, Bills DD, Sinnhuber RO. 1974. Accumulaiton of dietary polychlorinated biphenyls (Aroclor 1254) by rainbow trout (*Salmo gairdneri*). J Agric Good Chem 22:638-642.

Lillie RJ, Cecil HC, Bitman J, Fries GF, Verrett J. 1974. Differences in response of caged white leghorn layers to various polychlorinated biphenyls (PCBs) in the diet. Poult Sci 53:726-732.

Lillie RJ, Cecil HC, Bitman J, Fries GF, Verrett J. 1975. Toxicity of certain polychlorinated and polybrominated biphenyls on reproductive efficiency of caged chickens. Poult Sci 54:1550–1555.

Lincer JL. 1975. DDE-induced eggshell-thinning in the American kestrel: a comparison of the field situation and laboratory results. J Appl Ecol 12(3):781-793.

Lindsay DM, Sanders JG. 1990. Arsenic uptake and transfer in a simplified estuarine food chain. Environ Toxicol Chem 9:391-395.

Linzey AV. 1988. Effects of chronic polychlorinated biphenyls exposure on growth and reproduction of second generation white-footed mice (*Peromyscus leucopus*). Arch Environ Contam Toxicol 17:39-45.

Lipnick RL. 1993. Baseline toxicity QSAR models: A means to assess mechanism of toxicity for aquatic organisms and mammals. In: Gorsuch JW, Dwyer FJ, Ingersoll CG, Lapoint TW, eds, Environmental toxicology and risk assessment. Vol 2. STP 1216. American Society for Testing and Materials, Philadelphia, PA, pp. 610-619.

Lock RAC. 1975. Uptake of methylmercury by aquatic organisms from water and food. In: Koeman JH, Strik JJTWA, eds, Sublethal effects of toxic chemicals on aquatic organisms. Elsevier Press, Amsterdam. pp 61-79.

Long ER, Chapman PM. 1985. A sediment quality triad: measures of sediment contamination, toxicity and infaunal composition in Puget Sound. Mar Pollut Bull 16:405-415.

Long ER, MacDonald DD, Smith SL, Calder FD. 1995. Incidence of adverse biological effects within ranges of chemical concentrations in marine and estuarine sediments. Environ Manag 19:81-97.

Lopes TJ, Furlong ET. 2001. Occurrence and potential adverse effects of semivolatile organic compounds in streambed sediment, United States, 1992-1995. Environ Toxicol Chem 20(4):727-737.

Lowry LF, Frost KJ. 1981. Feeding and trophic relationships of phocid seals and walruses in the Eastern Bering Sea. In: Hood DW, Calder JA, eds, The eastern Bering Sea shelf: oceanography and resources. Vol 2. Department of Commerce, Washington, DC, pp 813-824.

Lundebye AK, Berntssen MHG, Wendelaar Bonga SE, Maage A. 1999. Biochemical and physiological responses in Atlantic salmon (*Salmo salar*) following dietary exposure to copper and cadmium. Mar Poll Bull 39:137-144.

Mac MJ, Schwartz TR. 1992. Investigations into the effects of PCB congeners on reproduction in lake trout from the Great Lakes. Chemosphere 25:189-192.

MacDonald DD, Carr RS, Calder FD, Long ER, Ingersoll CG. 1996. Development and evaluation of sediment quality guidelines for Florida coastal waters. Ecotoxicology 5:253-278.

MacDonald DD, Dipinto LM, Field J, Ingersoll CG, Long ER, Swartz RC. 2000. Development and evaluation of consensus-based sediment effect concentrations for polychlorinated biphenyls. Environ Toxicol Chem 19(5):1403-1413.

MacDonald JS, Birtwell IK, Kruuzynski GM. 1987. Food and habitat utilization by juvenile salmonids in the Campell River estuary. Can J Fish Aquat Sci 44: 1233-1246.

Macek KJ. 1968. Reproduction in brook trout (*Salvelinus fontinalis*) fed sublethal concentrations of DDT. J Fish Res Board Can 25:1787-1796.

Macek KJ, Korn S. 1970. Significance of the food chain in DDT accumulation by fish. J Fish Res Board Can 27:1496-1498.

Macek KJ, Rodgers CR, Stalling DL, Korn S. 1970. The uptake, distribution and elimination of dietary ^{14}C-DDT and ^{14}C-Dieldrin in rainbow trout. Trans Am Fish Soc 99:689-695.

Maher W, Butler E. 1988. Arsenic in the marine environment. Appl Organometallic Chem 2:191-214.

Malins DC, McCain BB, Brown DW, Sparks AK, Hodgins HO. 1980. Chemical contaminants and biological abnormalities in central and southern Puget Sound. NOAA Technical Memorandum OMPA-2. Environmental Conservation Division, National Marine Fisheries Service, Seattle, WA.

Malins DC, Mc Cain BB, Brown DW, Sparks AK, Hodgins HO, Chan S. 1982. Chemical contaminants and abnormalities in fish and invertebrates from Puget Sound. Environmental Conservation Division, National Marine Fisheries Service, Seattle, WA.

Malins DC, McCain BB, Brown DW, Chan SL, Myers MS, Landahl JT, Prohaska PG, Friedman AJ, Rhodes LD, Burrows DG, Gronlund WD, Hodgins HO. 1984. Chemical pollutants in sediments and diseases of bottom-dwelling fish in Puget Sound, Washington. Env Sci Tech 18:705-713.

Malins DC, Krahn MM, Brown DW, Rhodes LD, Myers MS, McCain BB, Chan S-L. 1985a. Toxic chemicals in marine sediment and biota from Mukilteo, Washington: Relationships with hepatic neoplasms and other hepatic lesions in English sole (*Parophrys vetulus*). J Nat Cancer Inst 74(2):487-494.

Malins DC, Krahn MM, Myers MS, Rhodes LD, Brown DW, Krone CA, McCain BB, Chan S-L. 1985b. Toxic chemicals in sediments and biota from a creosote-polluted harbor: Relationships with hepatic neoplasms and other hepatic lesions in English sole (*Parophrys vetulus*). Carcinogenesis 6(10):1463-1469.

Malins DC, McCain BB, Myers MS, Brown DW, Krahn MM, Roubal WT, Schiewe MH, Landahl JT, Chan S-L. 1987. Field and laboratory studies of the etiology of liver neoplasms in marine fish from Puget Sound. Environ Health Perspect 71:5-16.

Marine Mammal Center. 2000. California sea lion. Online circular at http://www.tmmc.org/csealion.htm. Updated June 2000; accessed May 9, 2001.

Mason CF. 1989. Water pollution and otter distribution: A review. Lutra 32:97-121.

Mason CF. 1993. Regional trends in PCB and pesticide contamination in northern Britain as determined in otter (*Lutra lutra*) scats. Chemosphere 26(5):941-944.

Mason CF. 1998. Decline in PCB levels in otters (*Lutra lutra*). Chemosphere 36:1969-1971.

Mason DF, O'Sullivan WM. 1992. Organochlorine pesticide residues and PCBs in otters (*Lutra lutra*) from Ireland. Bull Environ Contam Toxicol 48:387-393.

Mason CF, MacDonald SM. 1993. Impact of organochlorine pesticide residues and PCBs on otters (*Lutra lutra*) in eastern England. Sci Total Environ 138:147–160.

Mason CF, MacDonald SM. 1994. PCBs and organochlorine pesticide residues in otters (*Lutra lutra*) and in otter spraints from SW England and their likely impact on populations. Sci Tot Environ 144:305–312.

Mason CF, Madsen AB. 1993. Organochlorine pesticide residues and PCBs in Danish otters (*Lutra lutra*). Sci Tot Environ 133:73–81.

Mason CF, Macdonald SM, Bland HC, Ratford J. 1992. Organochlorine pesticide and PCB contents in otter (*Lutra lutra*) scats from western Scotland. Water Air Soil Pollut 64:617-626.

Mathisen J, Richards A. 1978. Status of great blue herons on the Chippewa National Forest. Loon 50:104-106.

Matsuda RI, Isaac GW, Dalseg RD. 1968. Fishes of the Green-Duwamish River. Water quality series no. 4. Municipality of Metropolitan Seattle, Seattle, WA.

Matta MB, Linse J, Cairncross C, Francendese L, Kocan RM. 2001. Reproductive and transgenerational effects of methylmercury or Aroclor 1268 on *Fundulus heteroclitus*. Environ Toxicol Chem 20:327-335.

Mauck WL, Mehrle PM, Mayer FL. 1978. effects of the polychlorinated biphenyl Aroclor 1254 on growth, survival and bone development in brook trout (*Salvelinus fontinalis*). J Fish Res Board Can 35:1084-1088.

Maurel C. 1997. Aquaporins and water permeability of plant membranes. Annu Rev Plant Physiol Mol Biol 48:399-429. In: Poschenrieder Ch, Barcelo J, 1999. Water relations in heavy metal stressed plants. In: Prasad MNV, Hagemeyer J, eds, Heavy metal stress in plants, from molecules to ecosystems. Springer-Verlag, Berlin, pp 207-229.

Maxson SJ, Oring LW. 1980. Breeding season time and energy budgets of the polyandrous spotted sandpiper. Behaviour 74:200-263.

Mayer FL, Mehrle PM, Sanders HO. 1977. Residue dynamics and biological effects of polychlorinated biphenyls in aquatic organisms. Arch Environ Contam Toxicol 5:501-511.

Mayer KS, Mayer FL, Witt A Jr. 1985. Waste transformer oil and PCB toxicity to rainbow trout. Trans Am Fish Soc 114:869-886.

McCain BB, Malins DC, Krahn MM, Brown DW, Gronlund WD, Moore LK, Chan S-L. 1990. Uptake of aromatic and chlorinated hydrocarbons by juvenile chinook salmon (*Oncorhynchus tshawytscha*) in an urban estuary. Arch Environ Contam Toxicol 19:10-16.

McCarty LS, Mackay D. 1993. Enhancing ecotoxicological modeling and assessment. Environ Sci Technol 27(9):1719-1728.

McEachran JD, Dunn KA. 1998. Phylogenetic analysis of skates, a morphologically conservative clade of elasmobranchs (Chondrichthyes: Rajidae). Copeia 1998(2):271-290.

McKim JM, GF Olson, GW Holcombe and EP Hunt. 1976. Long-term effects of methylmercuric chloride on three generation of brook trout (*Salvelinus fontinalis*): toxicity, accumulation, distributiom and elimination, J Fish Res Board Can 33:2726-2739.

McLane MAR, Hughes DL. 1980. Reproductive success of screech owls fed Aroclor® 1248. Arch Environ Contam Toxicol 9:661-665.

McLaren P, Ren P. 1994. Sediment transport in Elliott Bay and the Duwamish River, Seattle: Implications to estuarine management, GeoSea Consulting, Seattle, WA.

Meador JP. 1997. Comparative toxicokinetecs of tributyltin in five marine species and its utility in predicting bioaccumulation and acute toxicity. Aquat Toxicol 37:307-326.

Meador JP. 2000. An analysis in support of tissue and sediment based threshold concentrations of polychlorinated biphenyls (PCBs) to protect juvenile salmonids listed by the Endangered Species Act. Dated October 13, 2000.

Meador JP, Stein JE, Reichert WL, Varanasi U. 1995. A review of bioaccumulation of polycyclic aromatic hydrocarbons by marine organisms. Rev Environ Contam Toxicol 143:79-165.

Meador JP, Collier TK, Stein JE. 2002. Use of tissue and sediment-based threshold concentrations of polychlorinated biphenyls (PCBs) to protect juvenile salmonids listed under the US Endangered Species Act. Aquat Conserv: Marine Freshw Ecosyst 12:493-516.

Mehring AL, Brumbaugh JH, Sutherland AJ, Titus HW. 1960. The tolerance of growing chickens for dietary copper. Poult Sci 39:713-719.

Mehrle PM, Mayer FL. 1976. Di-2-ethylhexyl pthalate: residue dynamics and biological effects in rainbow trout. Proceedings, Annual conference of trace substances in environmental health, U-MO 519-524.

Melquist WE, Hornnocker MG. 1983. Ecology of river otters in west central Idaho. In: Kirkpatrick RL, ed, Wildlife monographs. Vol 83. The Wildlife Society, Bethesda, MD. 60 pp.

Melquist WE, Dronkert AE. 1987. River otter. In: Novak M, Bailer JA, Obbarel ME, eds, Wild furbearer management and conservation. University of Pittsburgh Press, Pittsburgh, PA, pp 627-641.

Metcalfe TL, Metcalfe CD. 1997. The trophodynamics of PCBs, including mono-and non-ortho congeners, in the food web of north-central Lake Ontario. Sci Total Environ 201:245-272.

Meyer JH, Pearce TA, Patlan SB. 1981. Distribution and food habits of juvenile salmonids in the Duwamish Estuary. Final report to Seattle District US Army Corps of Engineers. US Fish and Wildlife Service, Olympia, WA.

Michelsen TC, Bragdon-Cook K. 1993. Organic carbon normalization of sediment data: Technical information memorandum. Washington Department of Ecology, Olympia, WA.

Miles LJ, Parker GR. 1979. The effect of soil-added cadmium on several plant species. J Environ Qual 8(2):229-232.

Miller BS, Wingert RC, Borton SF. 1975. Ecological survey of demersal fishes in the Duwamish River and at West Point 1974. Report no. FRI-UW-7509. Fisheries Research Institute, University of Washington, Seattle, WA.

Miller BS, Wingert RC, Borton SF, Pierce KV, Griggs DT. 1977a. Ecological survey of demersal fishes in the Duwamish River and at West Point, 1975. Fisheries Research Institute, University of Washington, Seattle, WA.

Miller B, Simenstad A, Moulton L, Fresh KL, Funk FC, Karp WA, Borton SF. 1977b. Puget Sound baseline program nearshore fish survey. Fisheries Research Institute, College of Fisheries, University of Washington, Seattle, WA.

Miller DM, Stauffer GD. 1967. Study of the migration and spawning distribution runs of chinook and coho in the Green-Duwamish River system in the fall of 1965. Fisheries Research Institute, University of Washington, Seattle, WA.

Miller JE, Hassett JJ, Koeppe DE. 1977c. Interactions of lead and cadmium on metal uptake and growth of corn plants. J Environ Qual 6:18-20.

Miller JR, Miller JT. 1948. Nesting of the spotted sandpiper at Detroit, Michigan. Auk 65:558-567.

Miller PA, Lanno RP, McMaster ME, Dixon DG. 1993. relative contributions of dietary and waterborne copper to tissue copper burdens and waterborne-copper tolerance in rainbow trout (*Oncorhynchus mykiss*). Can J Fish Aquat Sci 5(8):1683-1689.

Mirenda RJ. 1986. Toxicity and accumulation of zinc in the crayfish, *Orconectes virilis* (Hagen). Bull Environ Contam Toxicol 37:387-394.

Moore JW, Ramamoorthy S. 1984. Organic chemicals in natural water. Springer-Verlag, New York, NY.

Morrow JE. 1980. The freshwater fishes of Alaska. Animal Resources Ecology Library, University of British Columbia, Vancouver, BC.

Mortimer MR, Miller GJ. 1994. Susceptibility of larval and juvenile instars of the sand crab, *Portunus pelagicus* (L.) to sea water contaminated by chromium, nickel or copper. Aust J Mar Freshwater Res 45:1107-1121.

Mowbray EE, Pursley D, Chapman JA. 1979. The status, population characteristics, and harvest of river otters in Maryland. Maryland Wildlife Administration. Waverly Press, Bethesda, MD.

Murai T, Andrews JW, Smith Jr RG. 1981. Effects of dietary copper on channel catfish. Aquaculture 22:353-357.

Muramoto S, Nishizaki H, Aoyama I. 1990. The critical levels and the maximum metal uptake for wheat and rice plants when applying metal oxides to soil. J Environ Sci Health, Part B 25:273-80.

Myers MS, Johnson LL, Olson OP, Stehr CM, Horness BF, Collier TK, McCain BB. 1998a. Toxicopathic hepatic lesions as biomarkers of chemical contaminant exposure and effects in marine bottomfish species from the Northeast and Pacific coasts. USA Mar Poll Bull 37:92-113.

Myers MS, Johnson LL, Hom T, Collier TK, Stein JE, U V. 1998b. Toxicopathic hepatic lesions in subadult English sole (*Pleuranectes vetulus*) from Puget Sound, Washington, USA: Relationships with other biomarkers of contaminant exposure. Mar Environ Res 45(1):47-67.

Nagy KA. 1987. Field metabolic rate and food requirement scaling in mammals and birds. Ecol Monogr 57: 111-128

Nagy KA, Girard IA, Brown TK. 1999. Energetics of free-ranging mammals, reptiles, and birds. Annu Rev Nutr 19:247-277.

Nebeker AV, Puglisi FA, DeFoe DL. 1974a. Effect of polychlorinated biphenyl compounds on survival and reproduction of the fathead minnow and flagfish. Trans Am Fish Soc 103:562-568.

Nebeker AV, Puglisi FA, DeFoe DL. 1974b. Effect of polychlorinated biphenyl compounds on survival and reproduction of the fathead minnow and flagfish. National Water Quality Laboratory, US Environmental Protection Agency, Duluth, MN.

Nebeker AV, Onjukha ST, Cairns MA. 1988. Chronic effects of contaminated sediment on *Daphnia magna* and *Chironomus tentans*. Bull Environ Contam Toxicol 41:574-581.

Nebraska Game and Parks Commission. 2000. The river otter. http://ngp.ngpc.state.ne.us/wildlife/otters.html. June 6, 2000.

Neff JM 1979. Polycyclic aromatic hydrocarbons in the aquatic environment. Sources, fates, and biological effects. Applied Science Publishers, London.

Nehlsen W, Williams JE, Lichatowich JA. 1991. Pacific salmon at the crossroads: Stocks at risk from California, Oregon, Idaho, and Washington. Fisheries 16 (2):4-21.

Nemec MD, Holson JF, Farr CH, Hood RD. 1998. Developmental toxicity assessment of arsenic acid in mice and rabbits. Reprod Toxicol 12(6):647-658.

Niimi AJ. 1983. Biological and toxicological effects of environmental contaminants in fish and their eggs. Can J Fish Aquat Sci 40:306-312.

Niimi AJ. 1996. Evaluation of PCBs and PCDD/Fs retention by aquatic organisms. Sci Total Environ 192:123-150.

Niimi AJ, Cho CY. 1983. Laboratory and field analysis of pentachlorophenol (PCP) accumulation by salmonids. Water Res 17: 1791-1795.

Niimi AJ, Lowe-Jinde L. 1984. Differential blood cell ratios of rainbow trout (*Salmo gairdneri*) exposed to methylmercury and chlorobenzenes. Arch Environ Contam Toxicol 13:303-311.

Nirmala K, Oshima Y, Lee R, Imada N, Honjo T, Kobayashi K. 1999. Transgenerational toxicity of tributyltin and its combined effects with polychlorinated biphenyls on reproductive processes in Japanese medaka (*Oryzias latipes*). Environ Toxicol Chem 18:717-721.

NMFS. 1997. Impacts of California sea lions and pacific harbor seals on salmonids and on the coastal ecosystems of Washington, Oregon, and California. NOAA Technical Memorandum NMFS-NWFSC-28. National Marine Fisheries Service, Seattle, WA.

NMFS. 2002. Unpublished data on PCB concentrations in juvenile chinook salmon captured in the Lower Duwamish Waterway during 1993 and 2000. Environmental Conservation Division, National Marine Fisheries Service, Seattle, WA.

NOAA. 1998. Factors contributing to the decline of chinook salmon: an addendum to the 1996 west coast steelhead factors for decline report. National Oceanic and Atmospheric Administration and National Marine Fisheries Service, Seattle, WA.

Norman DM. 2002a. Personal communication (email to Berit Bergquist, Windward Environmental LLC, regarding great blue herons in the LDW). Norman Wildlife Consulting, Shoreline, WA. April 5.

Norman DM. 2002b. Personal communication (telephone conversation with Berit Bergquist, Windward Environmental LLC, regarding spotted sandpiper in the LDW). Norman Wildlife Consulting, Shoreline, WA. March 29.

Norman DM. 2002c. Personal communication (email to Berit Bergquist, Windward Environmental LLC, regarding estimate of PCBs in heron egg subsamples). Norman Wildlife Consulting, Shoreline, WA. November 1.

Norman DM, Moul I, Breault A. 1989. Bald eagle incursions and predation in great blue heron colonies. Colonial Waterbirds 12:215-217.

Nosek, JA, Sullivan JR, Craven SR, Gendron-Fitzpatrick A, Peterson RE. 1993. Embryotoxicity of 2,3,7,8-tetrachlorodibenzo-p-dioxin in the ring-necked pheasant. Environ Toxicol Chem 12:1215-1222.

NYDEC. 1999. Technical guidance for screening contaminated sediments. Division of Fish, Wildlife and Marine Resources, New York State Department of Environmental Conservation, Albany, NY.

Nysewander DR, Evenson JR, Murphie BL, Cyra TA. 2001. Status and trends for a suite of key diving marine bird species characteristic of greater Puget Sound, as examined by the marine bird component, Puget Sound Ambient Monitoring Program (PSAMP). In: Puget Sound Research 2001: Abstracts & Biographies. Proceedings, meeting of the Puget Sound Water Quality Action Team, Bellevue, WA, February 12-14, 2001. Puget Sound Water Quality Action Team, Office of the Governor, Olympia, WA.

O'Connor JM, Huggett RJ. 1988. Aquatic pollution problems, north Atlantic coast, including Chesapeake Bay. Aquat Toxicol 11:163-190.

O'Neill SM, West JE, Hoeman JC. 1998. Spatial trends in the concentration of polychlorinated biphenyls (PCBs) in chinook (*Oncorhynchus tshawytscha*) and coho salmon (*O. kisutch*) in Puget Sound and factors affecting PCB accumulation: results from the Puget Sound Ambient Monitoring Program. In: Puget Sound Research '98 Proceedings, Puget Sound Water Quality Action Team, Olympia, WA. pp. 312-328.

O'Shea TJ, Stafford CJ. 1980. Phthalate plasticizers: accumulation and effects on weight and food consumption in captive starlings. Bull Environ Contam Toxicol 25:345-352.

Odenbro A, Kihlstrom JE. 1977. Frequency of pregnancy and ova implantation in triethyl lead-treated mice. Toxicol Appl Pharmacol 39:359-363.

Oehlmann J, Fioroni P, Stroben E, Markert B. 1996. Tributyltin (TBT) effects on *Ocinebrina aciculata* (Gastropoda: Muricidae): imposex development, sterilization, sex change and population decline. Sci Total Environ 188:205-223.

Oehlmann J, Bauer B, Minchin D, Schulte-Oehlmann U, Fioroni P, Markert B. 1998. Imposex in *Nucella lapillus* and intersex in *Littorina littorea*: interspecific comparison of two TBT-induced effects and their geographical uniformity. Hydrobiologia 318:199-213.

Oh SH, Nakaue H, Deagen JT, Whanger PD,Arscott GH. 1979. Accumulation and depletion of zinc in chick tissue metallothioneins. J Nutr 109: 1720-1729.

Oladimeji AA, Qadri SU, DeFreitas ASW. 1984. Long-term effects of arsenic accumulation in rainbow trout, *Salmo gairdneri*. Bull Environ Contam Toxicol 32:732-741

Oliver BG, Niimi AJ. 1983. Bioconcentration of chlorobenzenes from water by rainbow trout: correlations with partition coefficients and environmental residues. Environ Sci Technol 17:287-291.

Olson GF, Mount DI, Snarski, VM, Thorslund TW. 1975. Mercury residues in fathead minnows, *Pimephales promelas* Rafinesque, chronically exposed to methylmercury in water. Bull Environ Contam Toxicol 14:129-134.

Oring LW, Lank DB. 1986. Polyandry in spotted sandpipers: the impact of environment and experience. In: Rubenstein DI, Wrangham RW, eds, Ecological aspects of social evolution: Birds and mammals. pp 21-42.

Oring LW, Lank DB, Maxson SJ. 1983. Population studies of polyandrous sandpiper. Auk 100:272-285.

Overcash RM, Weber JB, Miles ML. 1982. Behavior of organic priority pollutants in the terrestrial system: Di-n-butyl phthalate ester, toluene, and 2,4 dinitro phenol. UNC-WRRI-82-171. Water Resources Research Institute, University of North Carolina, Chapel Hill, NC.

Page, LM, Burr BM. 1991. A field guide to freshwater fishes of North America north of Mexico. Houghton Mifflin, Boston, MA. 432 pp.

Pallson W. 2001. Personal communication (conversation with Windward Environmental LLC staff regarding English sole population dynamics in Elliott Bay). Washington Department of Fish and Wildlife, Olympia, WA.

Palm RC Jr. 1996. Specific humoral response of rainbow trout (*Oncorhynchus mykiss*) to injection, immersion, and oral immunization against *Vibrio anguillarum*. PhD thesis, University of Washington, Seattle, WA.

Palm RC Jr., Busch RA, Landolt ML. 1998. Route of vaccine administration: effects on the humoral response in rainbow trout (*Oncorhynchus mykiss*). Dis Aquat Org 33:157-166.

Palm RC Jr, Powell DB, Skillman A, Godtfredsen K. In prep. Immunocompetence of juvenile chinook salmon against *Listonella anguillarium* and growth following dietary exposure to a mixture of PAH compounds.

Panda KK, Lenka M, Panda BB. 1992. Monitoring and assessment of mercury pollution in the vicinity of a chloralkali plant. II Plant - availability, tissue-concentration and genotoxicity of mercury from agricultural soil contaminated with solid waste assessed in barley (*Hordeum vulgare* L.). Environ Pollut 76:33-42.

Parametrix. 1992. Bald eagle monitoring, 1989-1991. Prepared for METRO West Point Secondary Treatment Facilities. Parametrix, Inc., Kirkland, WA.

Patmont C. 1983. Water quality assessment of the Duwamish Estuary, Washington. Prepared for Municipality of Metropolitan Seattle. Harper-Owes Company, Seattle, WA.

Pattee OH. 1984. Eggshell thickness and reproduction in American kestrels exposed to chronic dietary lead. Arch Environ Contam Toxicol 13:29-34.

Patton JF, Dieter MP. 1980. Effects of petroleum hydrocarbons on hepatic function in the duck. Comp Biochem Physiol 65C:33-36.

Pauley GB, Bortz BM, Shepard MF. 1986. Species profiles: life histories and environmental requirements of coastal fishes and invertebrates (Pacific Northwest): Steelhead trout. Biological report 82 (11.82). TR EL-82-4. Prepared for US Fish and Wildlife Service and US Army Corps of Engineers. College of Ocean and Fishery Sciences, University of Washington, Seattle, WA.

Paulson D. 1993. Shorebirds of the Pacific Northwest. University of Washington Press, Seattle, WA.

Payne JF, Fancey LL, Rahimtula AD, Porter EL. 1987. Review and pespective on the use of mixed-runction oxygenase enzymes in biological monitoring. Comp Biochem Physiol C 86:233-245.

Payne PM, Selzer LA. 1989. The distribution, abundance and selected prey of the harbor seal, *Phoca vitulina concolor,* in southern New England. Mar Mammal Sci 5:173-192.

Peakall DB. 1974. Effects of di-N-butylphthalate and di-2-ethylhexylphthalate on the eggs of ring doves. Bull Environ Contam Toxicol 12:698-702.

Peakall DB, Lincer JL. 1972. Methyl mercury: its effect on eggshell thickness. Bull Environ Contam Toxicol 3(2):89-90.

Peakall DB, Peakall ML. 1973. Effect of a polychlorinated biphenyl on the reproduction of artificially and naturally incubated dove eggs. J Appl Ecol 10:863-868.

Peakall DB, Lincer JL, Bloom SE. 1972. Embryonic mortality and chromosomal alterations caused by Aroclor 1254 in ring doves. Environ Health Perspect 1:103-104.

Pearcy WG, Hancock D. 1978. Feeding habits of dover sole, *Microstomus pacificus;* rex sole, *Glyptocephalus zachirus;* slender sole, *Lyopsetta exilis;* and pacific sanddab, *Citharichthys sordidus,* in a region of diverse sediments and bathymetry off Oregon. Fish Bull 76(3):641-651.

Pearson TH, Rosenberg R. 1978. Macrobenthic succession in relation to organic enrichment and pollution of the marine environment. Oceanogr Mar Biol Ann Rev 16:229-311.

Penttinen OP, Kukkonen J. 1998. Chemical stress and metabolic rate in aquatic invertebrates: Threshold, dose-response relationships, and mode of toxic action. Environ Toxicol Chem 17(5):883-890.

Persaud D, Jaagumagi R, Hayton A. 1993. Guidelines for the protection and management of aquatic sediment quality in Ontario. ISBN 0-7729-9248-7. Ontario Ministry of Environment and Energy, Toronto, ON.

Phillips GR, Buhler DR. 1978. The relative contributions of methylmercury from food or water to rainbow trout (*Salmo gairdneri*) in a controlled laboratory environment. Trans Am Fish Soc 107:853-861.

Pillai MKK, Agarwal HC, Yadav DV. 1977. Tolerance, uptake and metabolism of DDT in *Gambusio affinis.* Indian J Exp Biol 15:40-41.

Pitcher KW. 1980. Food of the harbor seal, *Phoca vitulina richardsi,* in the Gulf of Alaska. US National Marine Fisheries Service Bulletin 78:544-549.

Pitcher KW, Calkins OG. 1979. Biology of the harbor seal, *Phoca vitulina richardsi,* in the Gulf of Alaska. Final. Outer Continental Shelf Environmental Assessment Program Research Unit 229. Bureau of Land Management, US Department of the Interior, Washington, DC.

Platonow N, Karstad C. 1973. Dietary effects of polychlorinated biphenyls on mink. Can J Comp Med 37:391-400.

Platonow NS, Reinhart BS. 1973. The effects of polychlorinated biphenyls (Aroclor 1254) on chicken egg production, fertility and hatchability. Can J Comp Med 37:341-346.

Poupoulis C, Jensen LS. 1976. Effect of high dietary copper on gizzard integrity of the chick. Poult Sci 55:113-121.

Powell DB, Palm RC Jr, Skillman A, Godtfredsen K. In press. Immunocompetence of juvenile chinook salmon against *Listonella anguillarum* following dietary exposure to Aroclor 1254.

Powell, DC, Aulerich RJ, Meadows JC, Tillitt DE, Powell JF, Restum JC, Stromborg KL, Giesy JP, Bursian SJ. 1997. Effects of 3,3',4,4',5-pentachlorobiphenyl (PCB 126), 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD), or an extract derived from field-collected cormorant eggs injected into double-crested cormorant (*Phalacrocorax auritus*) eggs. Environ Toxicol Chem 16(7):1450-1455.

PSDDA. 1988. PSDDA Reports - Final environmental impact statement - unconfined open-water disposal sites for dredged material, phase I (Central Puget Sound). Prepared for Puget Sound Dredged Disposal Authority. US Army Corps of Engineers, Seattle, WA.

PSWQA. 1990. Managing non-point pollution. Puget Sound Water Quality Authority, Seattle, WA.

PTI. 1988. Sediment quality values refinement: Volume I. Update and evaluation of Puget Sound AET. Prepared for Puget Sound Estuary Program (PSEP), US Environmental Protection Agency Region 10. PTI Environmental Services, Inc., Bellevue, WA.

PTI. 1990. Duwamish channel and settling basin sediment bioassay. Prepared for the US Army Corps of Engineers. PTI Environmental Services, Inc., Bellevue, WA.

PTI. 1991. Reference area performance standards for Puget Sound. Final report prepared for US Environmental Protection Agency, Region 10, Seattle, WA and Washington Department of Ecology, Olympia, WA. PTI Environmental Services, Inc., Bellevue, WA.

PTI. 1996. Proposed dredging of Slip No. 4, Duwamish River, Seattle, WA. Prepared for Crowley Marine Services. PTI Environmental Services, Inc., Bellevue, WA.

Restum JC, Bursian SJ, Giesy JP, Render JA, Helferich WG, Shipp EB, Verbrugge DA. 1998. Multigenerational study of the effects of consumption of PCB-contaminated carp from Saginaw Bay, Lake Huron, on mink. 1. Effects on mink reproduction, kit growth and survival, and selected biological parameters. J Toxicol Environ Health 54(A):343-375.

Rhead MM, Perkins JM. 1984. An evaluation of the relative importance of food and water as sources of p,p'-DDT to the goldfish *Carassius auratus* (L.). Water Res 18:719-725.

Rhodes LD, Myers MS, Gronlund WD, McCain BB. 1987. Epizootic characteristics of hepatic and renal lesions in English Sole, *Parophrys vetulus*, from Puget Sound. J Fish Biol 31:395-407.

Rice CA, Myers MS, Willis ML, French BL, Casillas E. 2000. From sediment bioassay to fish biomarker - connecting the dots using simple trophic relationships. Mar Environ Res 50:527-533.

Rice CP, O'Keefe P. 1995. Sources, pathways, and effects of PCBs, dioxins, and dibenzofurans. In: Hoffman DJ, Rattner BA, Burton GA Jr, Cairns, J Jr, eds, Handbook of ecotoxicology. Lewis Publishers, Boca Raton, FL, pp 424-468.

Rice SD, Short JW, Stickle WB. 1989. Uptake and catabolism of tributyltin by blue crabs fed TBT contaminated prey. Mar Environ Res 27:137-145.

Ridgeway LP, Karnofsky DA. 1952. The effects of metals on the chick embryo: Toxicity and production of abnormalities in development. Ann NY Acad Sci 55:203-215.

Rieman BE, McIntyre JD. 1993. Demographic and habitat requirements for conservation of bull trout. USFS Publication IN-GTR-302:1-38. US Fish and Wildlife Service, Washington, DC.

Risebrough RW, Anderson DW. 1975. Some effects of DDE and PCB on mallards and their eggs. J Wildl Manag 39(3):508-513.

Roberson RH, Schaible PJ. 1960. The tolerance of growing chicks for high levels of different forms of zinc. Poult Sci 39:893-896.

Robertson W. 2002. Personal communication (conversation with Matt Luxon regarding fish surveys in LDW). Diver, Seattle Aquarium, Seattle, WA. May 21, 2002.

Rodgers DW, Beamish FWH. 1982. Dynamics of dietary methylmercury in rainbow trout, *Salmo gairdneri*. Aquat Toxicol 2:271-290.

Roffe TJ, Mate BR. 1984. Abundances and feeding habits of pinnipeds in the Rogue River, Oregon. J Wildl Manage 48: 1262-1274.

Rolfe GK, Bazzaz FA. 1975. Effect of lead contamination on transpiration and photosynthesis of loblolly pine and autumn olive. Forest Sci 21:33-35.

Roose P, Brinkman UAT. 2000. Volatile organic compounds in various marine organisms from the southern North Sea. Mar Poll Bull 40(12):1167-1177.

Ross P, DeSwart R, Addison R, VanLoveren H, Vos J, Osterhaus A. 1996. Contaminant-induced immunotoxicity in harbour seals: wildlife at risk? Toxicology 112:157–169.

Ruggerone, G. 2002. Personal communication (various telephone conversations and e-mails with Kathy Godtfredsen, Windward Environmental). Biologist, Natural Resource Consulting, Inc., Seattle, WA.

Safe S. 1984. Polychlorinated biphenyls (PCBs) and polybrominated biphenyls (PBBs): biochemistry, toxicology, and mechanism of action. CRC Crit Rev Toxicol 13:319-395.

Safe S. 1990. Polychlorinated biphenyls (PCBs), dibenzo-*p*-dioxins (PCDDs), dibenzofurans (PCDFs), and related compounds: environmental and mechanistic considerations which support the development of toxic equivalency factors (TEFs). Crit Rev Toxicol 21(1):51-88.

Safe S. 1991. Polychlorinated dibenzo-*p*-dioxins and related compounds: sources, environmental distribution and risk assessment. Environ Carcin Ecotoxicol Rev C9(2):261-302.

Safe S. 1992. Toxicology, structure—function relationship, and human and environmental health impacts of polychlorinated biphenyls: progress and problems. Environ Health Perspect 100:259-268.

Safe SH. 1994. Polychlorinated biphenyls (PCBs): environmental impact, biochemical and toxic responses, and implications for risk assessment. Crit Rev Toxicol 24(2):87-149.

Sager DB, Girard DM. 1994. Long-term effects on reproductive parameters in female rats after translactational exposure to PCBs. Environ Res 66:52-76.

SAIC. 1992. PSDDA bioassays for Duwamish Channel sediments: bioassay data. Prepared for US Army Corps of Engineers. Science Applications International Corporation, Bothell, WA.

Sample BE, Opresko DM, Suter GW. 1996. Toxicological benchmarks for wildlife. 1996 revision. ES/ERM-86/R3. Office of Environmental Management, US Department of Energy, Washington, DC.

Sato M. 1997. The price of taming a river. The decline of Puget Sound's Green/Duwamish Waterway. The Mountaineers, Seattle, WA.

Schaffer KE. 1989. Seasonal and size variations in diets of harbor seals. In: 8th Biennial Conference on the Biology of Marine Mammals, December 7-11, 1989, Pacific Groves, CA, p.62.

Scheuhammer AM. 1988. Chronic dietary toxicity of methylmercury in the zebra finch, *Poephila guttata*. Bull Environ Contam Toxicol 40:123-130.

Schiewe MH, Weber DD, Myers MS, Jacques FJ, Reichert WL, Krone CA, Malins DC, McCain BB, Chan SL, Varanasi U. 1991. Induction of foci of cellular alteration and other hepatic lesions in English sole (*Paraphrys vetulus*) exposed to an extract of an urban marine sediment. Can J Fish Aquat Sci 48.

Schimmel SC, Patrick JM Jr, Forrester J. 1976. Heptachlor: Uptake, depuration, retention, and metabolism by spot, *Leiostomus xanthurus*. Toxicol Environ Health 2:169-178.

Schimmel SC, Patrick JM Jr, Forrester J. 1977. Uptake and toxicity of toxaphene in several estuarine organisms. Arch Environ Contam Toxicol 5:353-367.

Schlatterer B, Coenen TMM, Ebert E, Grau R, Hilbig V, Munk R. 1993. Effects of Bis(tri-*n*-butyltin)oxide in Japanese quail exposed during egg laying period: an interlaboratory comparison study. Arch Environ Contam Toxicol 24:440-448.

Schmitt CJ, Dethloff GM, eds. 2000. Biomonitoring of environmental status and trends (BEST) program: selected methods for monitoring chemical contaminants and their effects in aquatic ecosystems. Information and Technology Report USGS/BRD-2000—0005. Biological Resources Division, US Geological Survey, Columbia, MO.

Schock K, Zhong J, Shuman R, Munger S. 1998. Simulating water quality in the Duwamish Estuary and Elliott Bay: Comparing effects of CSOs and other sources. In: Puget Sound Research '98 Proceedings, Puget Sound Water Quality Action Team, Olympia, WA.

Schrankel KR, Kreamer BL, Hsia MTS, 1982. Embryotoxicity of 3,3',4,4'-tetra chloroazoxybenzene in the chick embryo. Arch Environ Contam Toxicol 11:195-202.

Schroeder HA, Mitchener M. 1971. Toxic effects of trace elements on the reproduction of mice and rats. Arch Environ Health 23:102-106.

Schultz EA, Tiffarny JB. 1965. Effects of density difference on estuarine hydraulics. Chapter V. In: Wicker CF, ed, Report No. 3, Evaluation of present state of knowledge factors affecting tidal hydraulics and related phenomena. Committee On Tidal Hydraulics. US Army Corps of Engineers (as cited in Curl et al. 1987).

Scott ML. 1977. Effects of PCBs, DDT, and mercury compounds in chickens and Japanese quail. Federation Proceedings 36:1888-1893.

Scott WB, Crossman EJ. 1973. Freshwater fishes of Canada. (Reprinted 1990). Bull. Fish Res Board Can (184). 966 pp.

Scott ML, Vadehra DV, Mullenhoff PA, Rumsey GL, Rice RW. 1971. Results of experiments on the effects of PCBs on laying hen performance. In: Proceedings, 1971 Cornell Nutrition Conference for Feed Manufacturers. Agricultural Experiment Station, Departments of Animal and Poultry Science, Cornell University, Ithaca, NY, pp. 56-64.

Seegal RF. 1996. Epidemiological and laboratory evidence of PCB-induced neurotoxicity. Crit Rev Toxicol 26(6):709-737.

Seuss MJ. 1976. Science of the total environment. Volume 6, pp 239-250.

Shacklette HT, Erdman JA, Harms TF, Papp SE. 1978. Trace elements in plant foodstuffs. In: Oehme F, ed, Toxicity of heavy metals in the environment. Part I. Marcel Dekker, New York, NY, pp 25-68.

Shannon J. 2001. Personal communication (telephone conversation with Matt Luxon, Windward Environmental LLC, Seattle, WA, regarding bull trout captured in the lower Duwamish River). Fisheries Biologist, Taylor and Associates, Seattle, WA. April 11.

Sharma S, Kanwar KC. 1985. Reproductive performance in mice following lead administration. Res Bull Panjab Univ 36:389-394

Shimizu A, Kimura S. 1987. Effect of bis(tributyltin)oxide on gonadal development of a salt-water goby, *Chasmichtyhys dolichognathus*: Exposure during mating period. Bull Todai Reg Fish Res Lab 123:45-49.

Shubat PJ, Curtis LR. 1986. Ration and toxicant preexposure influence dieldrin accumulation by rainbow trout (*Salmo gairdneri*). Environ Toxicol Chem 5:69-77.

Shugart L, Bickman J, Jackim G, McMahon G, Ridley W, Stein J, Steinert S. 1992. DNA Alterations. In: Biomarkers: biochemical physiological and histological markers of anthropogenic stress. Lewis Publishers, Boca Raton, FL, pp 125-153.

Silberhorn EM, Glauert HP, Robertson LW. 1990. Carcinogenicity of polyhalogenated biphenyls: PCBs and PBBs. Crit Rev Toxicol 20(6):397-426.

Sivarajah K, Franklin, Williams WP. 1978. The effects of polychlorinated biphenyls on plasma steroid levels and hepatic microsomal enzymes in fish. J Fish Biol 13:401-409.

Sjöåsen T, Ozolins J, Greyerz E, Olsson M. 1997. The otter (*Lutra lutra*) situation in Latvia and Sweden related to PCB and DDT levels. Ambio 26:196-201.

Slaga TJ, Bracken WM, Viaje A, Berry DL, Fischer SM, Miller DR, Levin W, Convey AH, Yagi H, Jerina DM. 1978. Tumor initiating and promoting activities of various benzo(a)pyrene metabolites in mouse skin. In: Jones PW, Freudenthal RI, eds, Carcinogenesis. A comprehensive survey. Vol 3. Polynuclear aromatic hydrocarbons: Second international symposium on analysis, chemistry, and biology. Raven Press, New York, NY, pp. 371-392.

Smith MS. 1969. Responses of chicks to dietary supplements of copper sulphate. Brit Poult Sci 10:97-108.

Smith RM, Cole CF. 1973. Effects of egg concentrations of DDT and dieldrin on development in winter flounder (*Pseudopleuronectes americanus*). J Fish Res Board Can 30:1894-1898.

Smith RT. 1936. Report on the Puget Sound otter trawl investigations. Wash Dep. Fish. Rep 36. Washington Department of Fisheries, Olympia, WA.

Smith SL, MacDonald DD, Keenleyside KA, Ingersoll CG, Field LJ. 1996. A preliminary evaluation of sediment quality assessment values for freshwater ecosystems. J Great Lakes Res 22:624-638.

Snarski VM, Olson GF. 1982. Chronic toxicity and bioaccumulation of mercuric chloride in the fathead minnow (*Pimehales promelas*). Aquat Toxicol 2:143-156.

Snoeij NJ, Pennicks AH, Seinen W. 1985. Biological activity of organotonin compounds—an overview. Environ Res 44:335-353.

Snyder NF, Wiley JW. 1976. Sexual size dimorphism in hawks and owls of North America. Ornithol Monogr 20.

Southworth GR, Beauchamp JJ, Schmieder PK. 1978. Water research. Volume 12, pp 973-977.

Spalding MG, Frederick PC, McGill HC, Bouton SN, McDowell LR. 2000. Methylmercury accumulation in tissues and its effects on growth and appetite in captive great egrets. J Wildl Diseas 36(3):411-422.

Spann JW, Heinz GH, Camardese B, Hill EF, Moore JF, Murray HC. 1986. Differences in mortality among bobwhite fed methylmercury chloride dissolved in various carriers. Environ Toxicol Chem 5:721-724.

Spearman JW. 1991a. Sediment sampling and analysis: South Park marina. Jay W Spearman, Consulting Engineer, Kirkland, WA.

Spearman JW. 1991b. Sediment sampling and analysis: Brown and Morton properties, Duwamish Waterway. Jay W Spearman, Consulting Engineer, Kirkland, WA.

Spearman JW. 1999. Sediment sampling and analysis, James Hardie Gypsum Inc., Duwamish Waterway, Seattle, Washington. Jay W. Spearman, Consulting Engineer, Kirkland, WA.

Spehar RL, Nelson HP, Swanson MJ, Renoos JW. 1985. Pentachlorophenol toxicity to amphipods and fathead minnows at different pH test values. Environ Tox Chem 4:389-397.

Spinola RM, Serfass TL, Brooks RP. 1999. Radiotelemetry study: river otters reintroduction at Letchworth State Park. Progress report no. 1. New York State Department of Environmental Conservation, New York River Otter Project, and New York State Office of Parks, Recreation, and Historic Preservation, Albany, NY.

Stahl JL, Greger JL, Cook ME. 1990. Breeding-hen and progeny performance when hens are fed excessive dietary zinc. Poult Sci 69:259-263.

Stalmaster, M. 1987. The bald eagle. Universe Books. New York, NY. 227 pp.

Stalmaster MV, Gessaman JA. 1982. Food consumption and energy requirements of captive bald eagles. J Wildl Manage 46:646-654.

Staples CA, Adams WJ, Parkerton TF, Gorsuch JW, Biddinger GR, Reinert KH. 1997. Aquatic toxicity of eighteen phthalate esters. Environ Toxicol Chem 16: 875-891.

Stein JE, Collier TK, Reichaer WL, Casillas E, Hom T, Varanasi U. 1992. Bioindicators of contaminant exposure and sublethal effects: studies with benthic fish in Puget Sound, Washington. Environ Toxicol Chem 11:701-714.

Stein JE, Hom T, Collier TK, Brown DW, Varanasi U. 1995. Contaminant exposure and biochemical effects in outmigrant juvenile chinook salmon from urban and non-urban estuaries of Puget Sound, Washington. Environ Toxicol Chem 14(6):1019-1029.

Stenson GB, Badgero GA, Fisher HD. 1984. Food habits of the river otter *Lutra canadensis* in the marine environment of British Columbia. Can J Zool 62:88-91.

Stevens Thompson & Runyan. 1972. Effect of dredging on water quality and sediment transport in the Duwamish Estuary for the Corps of Engineers. Stevens, Thompson & Runyan, Inc., Seattle, WA.

Stewart BS, Leatherwood SL, Yochem PK. 1989. Harbor seal tracking and telemetry by satellite. Mar Mam Sci 5:361-375.

Stoewsand GS, Anderson JA, Gutenmann WH, Bache CA, Lisk DJ. 1971. Eggshell thinning in Japanese quail fed mercuric chloride. Science 173:1030-1031.

Strand J. 1999a. Personal observation. In: King County combined sewer overflow water quality assessment for the Duwamish River and Elliott Bay. Appendix B2, B3, & B4: Human Health, Wildlife, and aquatic life risk assessments. King County Department of Natural Resources, Seattle, WA.

Strand J. 1999b. Personal observation. In: King County combined sewer overflow water quality assessment for the Duwamish River and Elliott Bay. Appendix C: Issue papers. King County Department of Natural Resources, Seattle, WA.

Strek JH, Weber JB. 1980. Absorption and translocation of polychlorinated biphenyls (PCBs) by weeds. Proc South Weed Sci Soc. 33:226-232.

Strek JH, Weber JB. 1982. Adsorption and reduction in bioactivity of polychlorinated biphenyl (Aroclor 1254) to redroot pigweed by soil organic matter and montmorillonite clay. Soil Sci Soc Am J 46:318-22.

Striplin. 1996a. PSDDA chemical characterization of Duwamish Waterway and upper turning basin FY97 operations and maintenance dredging. Prepared for US Army Corps of Engineers. Striplin Environmental Associates Inc., Olympia, WA.

Striplin. 1996b. Development of reference value ranges for benthic infauna assessment endpoints in Puget Sound. Prepared for the Washington State Department of Ecology, Lacey, WA. Striplin Environmental Associates, Olympia, WA.

Striplin. 1998. Benthic infaunal communities in the vicinity of the Duwamish Diagonal combined sewer overflow. Final report. Prepared for King County Water Quality Division, Seattle WA. Striplin Environmental Associates, Olympia, WA.

Sturm A, da Silva de Assis HC, Hansen P-D. 1999. Cholinesterases of marine teleost fish: Enzymological characterization and potential use in the monitoring of neurotoxic contamination. Mar Environ Res 47:389-398.

Suzuki, T. 1979. Dose-effect and dose-response relationships of mercury and its derivatives. In: Nriagu JO, ed, The biogeochemistry of mercury in the environment. Elsevier/North-Holland Biomedical Press, New York, NY, pp. 399-431.

Swarth C. 2002. California's wildlife: spotted sandpiper. Wildlife and Habitat Data Analysis Branch, California Department of Fish and Game. February 8, 2002. http://www.dfg.ca.gov/whdab/cwhr/B170.html.

Swartz RC. 1999. Consensus sediment quality guidelines for polycyclic aromatic hydrocarbon mixtures. Environ Toxicol Chem 18(4):780-787.

Sweeney SJ, Neiman KE, Strong TR, Artman VL. 1992. West Point secondary treatment facilities bald eagle monitoring summary report for 1989-1991 nesting seasons. Prepared for Metro. Parametrix, Inc., Bellevue, WA.

Tabor JE, Wight HM 1977. Population status of river otter in western Oregon. J Wildl Manag 41:692-699.

Tagatz ME, Plaia GR, Deans CH. 1986. Toxicity of dibutyl phthalate-contaminated sediment to laboratory- and field-colonized estuarine benthic communities. Bull Environ Contam Toxicol 37: 141-150.

Takeda H, Shimma Y. 1977. Effects of toxic amounts of dietary zinc on the growth and body components of rainbow trout at two levels of calcium. Bull Fresh Fish Res 27:103-109.

Tanner CD. 1991. Potential intertidal habitat restoration sites in the Duwamish River Estuary. EPA 910/9-91-050. US Environmental Protection Agency, Region 10, Seattle, WA.

Tans M, Hugla JL, Libois RM, Rosoux R, Thome JP. 1996. Contamination of European otters (*Lutra lutra*) by PCB congeners and organochlorinated pesticides in the wetlands of western France. Neth J Zool 46(3-4):326-336.

Tas JW, Keizer A, Opperhuizen A. 1993. Bioaccumulation and lethal body burden of four trioganotin compounds [dissertation]. In: fate and effects of triorganotins in the aqueous environment. University of Utrecht, Utrecht, Netherlands, pp 117-132.

Taylor WJ, Shreffler DK, Cordell JR. 1999. Duwamish East Waterway channel deepening project: alternative dredge disposal sites juvenile salmonid and epibenthic prey assessment. Technical report. Preliminary draft. Prepared for Port of Seattle. Taylor Associates, Seattle, WA.

Terres JK. 1987. The Audubon Society encyclopedia of North American birds. Alfred A. Knopf. Inc., New York, NY. 1109 p.

Tetra Tech. 1988. Elliott Bay action program: analysis of toxic problem areas. Prepared for Puget Sound Estuary Program (PSEP), US Environmental Protection Agency, Region 10. Tetra Tech, Inc., Bellevue, WA.

Tetra Tech. 1996. Contaminant ecology of fish and wildlife of the lower Columbia River. Lower Columbia River Bi-State Program, draft report. TC 0941-01. Tetra Tech, Inc., Seattle, WA.

Thomann RV, Komlos J. 1999. Model of biota-sediment accumulation factor for polycyclic aromatic hydrocarbons. Environ Toxicol Chem 18:1060-1068.

Thomann RV, Mahony JD, Mueller R. 1995. Steady-state model of biota sediment accumulation factor for metals in two marine bivalves. Environ Toxicol Chem 14(11): 1989-1998.

Thomas P. 1988. Reproductive endocrine function in female Atlantic croaker exposed to pollutants. Mar Environ Res. 24:179-183.

Thompson, JAJ, Sheffer MG, Pierce RC, Chau YK, Cooney JJ, Cullen WR, Maguire RJ. 1985. Organotin compounds in the aquatic environment: Scientific criteria for assessing their effects on environmental quality. Publ NRCC 22494. National Research Council of Canada, Ottawa, ON. 284 pp.

Tillitt DE, Ankley GT, Giesy JP, Ludwig JP, Matsuba HK, Weseloh DV, Ross PS, Bishop CA, Sileo L, Stromberg KL, Larson J, Kubiak TJ. 1992. Polychlorinated biphenyl residues and egg mortality in double-crested cormorants from the Great Lakes. Environ Tox Chem 11:1281-1288.

Tillitt DE, Gale RW, Meadows JC, Zajicek JL, Peterman PH, Heaton SN, Jones PD, Bursian SJ, Kubiak TJ, Giesy JP, Aulerich RJ. 1996. Dietary exposure of mink to carp from Saginaw Bay. 3. Characterization of dietary exposure to planar halogenated hydrocarbons, dioxin equivalents, and biomagnification. Environ Sci Technol 30:283-291.

Tilson HA, Jacobson JL, Rogan WJ. 1990. Polychlorinated biphenyls and the developing nervous system: Cross-species comparisons. Neurotoxicol Teratol 12(3):239–248.

Tokranov AM, Maksimenkov VV. 1995. Feeding habits of predatory fishes in the Bol'shaya River estuary (West Kamchatka). J. Ichthyol 35(9):102-112.

Toweill DE 1974. Winter food habits of river otters in western Oregon. J Wildl Manag 38(1):107-111

Truelove JD, Grant D, Mes J, Tryphonas H, Trypohonas L, Zawidzka Z. 1982. Polychlorinated biphenyl toxicity in the pregnant cynomolgus monkey: A pilot study. Arch Environ Contam Toxicol 11:583-588.

Tsuda T, Aoki S, Kojima M, Harada H. 1990a. The influence of pH on the accumulation of tri-n-butyltin chloride and triphenyltin chloride in carp. Comp Biochem Physiol 95C:151-153.

Tsuda T, Aoki S, Kojima M, Harada H. 1990b. Differences between freshwater and seawater-acclimated guppies in the accumulation and excretion of tri-n-butyltin chloride and triphenyltin chloride. Water Res 24:1373-1376.

Tumasonis CF, Bush B, Baker FD. 1973. PCB levels in egg yolks associated with embryonic mortality and deformity of hatched chicks. Arch Environ Contam Toxicol 1(4):312-324.

USFWS. 2000. GIS coverage of Duwamish intertidal habitats. Contact: Carol Langston, GIS Analyst, Curtis Tanner, Fish and Wildlife Biologist. US Fish and Wildlife Service, Lacey, WA.

van der Weiden MEJ, Bleumink R, Seinen W, van den Berg M. 1994. Concurrence of P450 1A induction and toxic effects in the mirror carp (*Cyprinus carpio*), after administration of a low dose of 2,3,7,8-tetrachlorodibenzo-p-dioxin. Aquat Toxicol 29:147-162.

Van Loveren H, Ross PS, Osterhaus ADME, Vos JG. 2000. Contaminant-induced immunosuppression and mass mortalities among harbor seals. Toxicol Lett 112/113:319-324.

Van Luik A. 1984. Mined land reclamation using polluted urban navigable waterway sediments. II: Organics. J Environ Qual 13(3):415-422.

Van Wezel AP, De Vries DAM, Kostense S, Sijm DTHM, Oppenhuizen A. 1995. Intraspecies variation in lethal body burdens of narcotic compounds. Aquat Toxicol 33:325-342.

Van den Berg M, Birnbaum L, Bosveld ATC, Brunstrom B, Cook P, Feeley M, Giesy JP, Hanberg A, Hasegawa R, Kennedy SW, Kubiak T, Larsen JC, van Leeuwen FX, Liem AK, Nolt C, Peterson RE, Poellinger L, Safe S, Schrenk D, Tillitt D, Tysklind M, Younes M, Waern F, Zacharewski T. 1998. Toxic equivalency factors (TEFs) for PCBs, PCDDs, PCDFs for humans and wildlife. Environ Health Perspect 106:775-792.

Vangronsveld J, Clijsters H. 1994. Toxic effects of metals. Department SBG, Limburgs Universitair, Diepenbeek, Belgium.

Varanasi U. 1989. Metabolism of polycyclic aromatic hydrocarbons in the aquatic environment. CRC Press, Boca Raton, FL.

Varanasi U, Stein JE, Nishimoto M. 1989. Biotransformation and disposition of polycyclic aromatic hydrocarbons (PAH) in fish. In: Varanasi U, ed, Metabolism of polycyclic aromatic hydrocarbons in the aquatic environment. CRC Press Inc, Boca Raton, FL. p 93.

Varanasi U, Stein JE, Johnson LL, Collier TK, Casillas E, Myers MS. 1992. Evaluation of bioindicators of contaminant exposure and effects in coastal ecosystems. In: McKenzie DH, Hyatt DE, McDonald VJ, eds, Ecological indicators. Vol. 1. Proceedings of an international symposium, Ft Lauderdale, FL.

Varanasi U, Casillas E, Arkoosh MR, Misitano D, Stein JE, Hom T, Collier TK, Brown DW. 1993. Contaminant exposure and associated biochemical effects in outmigrant juvenile chinook salmon (*Onchorhynchus tschawytscha*) from urban and nonurban estuaries of Puget Sound, Washington. NOAA technical memorandum NMFS-FWFSC-8. Northwest Fisheries Science Center, National Marine Fisheries Service, Seattle, WA.

Vos JG. 1977. Immune suppression as related to toxicology. Crit Rev Toxicol 5(1):67-101.

Walker MK, Spitsbergen JM, Olson JR, Peterson RE. 1991. 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) toxicity during early life stage development of Lake Trout (*Salvelinus namaycush*). Can J Fish Aquat Sci 48:875-883.

Wallnofer PR, Engelhardt G. 1984. In: Hock B, Elstner EF, eds, Pflanzentoxicologie. Bibliographisches Institut, Mannheim. pp 95-117.

Walsh AR, O'Halloran J, Gower AM. 1994. Some effects of elevated levels of chromium (III) in sediments to the mullet *Chelon labrosus* (R). Ecotoxicol Environ Safety 27:168-176.

Ward GS, Cramm GC, Parrish PR, Trachman H, Slesinger A. 1981. Bioaccumulation and chronic toxicity of bis(tributyltin)oxide (TBTO): tests with a saltwater fish. In: Branson DR, Dickson KL, eds, Aquatic Toxicology And Hazard Assessment, Fourth Conference. STP 737. American Society for Testing and Materials, Philadelphia PA, pp 183-200.

Warlen SM, Wolfe DA, Lewis CW, Colby DR. 1977. Accumulation and retention of dietary ^{14}C-DDT by Atlantic menhaden. Trans Am Fish Soc 106:95-104.

Warner EJ, Fritz RL. 1995. The distribution and growth of Green River Chinook salmon and chum salmon outmigrants in the Duwamish Estuary as a function of water quality and substrate. Muckleshoot Indian Tribe. Auburn, WA.

Watson JW, Garrett MG, Anthony RG. 1991. Foraging ecology of bald eagles in the Columbia River estuary. J Wildl Manage 55(3):492-499.

Watson JW, Pierce DJ. 1998. Migration, diets, and home ranges of bald eagles breeding along Hood Canal and at Indian Island, Washington. WDFW 525. Wildlife Management Department, Washington Department of Fish and Wildlife, Olympia, WA.

Watson JW, Pierce DJ. 2001. Skagit River bald eagles: Movements, origins and breeding population status. WDFW 727. Wildlife Management Department, Washington Department of Fish and Wildlife, Olympia, WA.

Watson JW, Mundy D, Begley JS, Pierce DJ. 1995. Responses of nesting bald eagles to the harvest of geoduck clams (*Panopea abrupta*). Washington Department of Fish and Wildlife, Olympia, WA.

WDFW. 1993. 1992 Washington state salmon and steelhead stock inventory. Washington Department of Fisheries, Washington Department of Wildlife, and Western Washington Treaty Indian Tribes, Olympia, WA.

WDFW. 1999. Monitoring of 1998-99 California sea lion predation in the Lake Washington Estuary and the Lower Duwamish River. Washington Department of Fish and Wildlife, Olympia, Washington.

WDFW 2000. Streamnet GIS. Bull Trout Status/Distribution table: bulltrout.evt.e00. Created 7/21/00. Data Manager: Martin Hudson (360) 902-2487.

WDFW 2003. Species of concern in Washington State. Current through May 2003. http://www.wa.gov/wdfw/wlm/diversty/soc/soc.htm.

WDOE. 1994. Washington state pesticide monitoring program. 1993 surface water sampling report. Publication No. 94-164. Washington Department of Ecology, Olympia, WA.

Weber JB, Mrozek E Jr. 1979. Polychlorinated biphenyls: Phytotoxicity, absorption and translocation by plants, and inactivation by activated carbon. Bull Environ Contam Toxicol 23:412-17.

Weeks BA, Warinner JE. 1984. Effects of toxic chemicals on macrophage phagocytosis in two estuarine fishes. Mar Environ Res 14:327-335.

Weeks BA, Warinner JE. 1986. Functional evaluation of macrophages in fish from a polluted estuary. Vet Immunol Immunopathol 12:313-320.

Weeks BA, Warinner JE, Mason PL, McGinnis DS. 1986. Influence of toxic chemicals on the chemotactic response of fish macrophages. J Fish Biol 28:653-658.

Weitkamp DE, Campbell RF. 1980. Port of Seattle Terminal 107 fisheries study. Parametrix, Inc., Bellevue, WA.

Weitkamp DE, Schadt TH. 1982. 1980 juvenile salmonid study. 82-0415-012F. Prepared for Port of Seattle. Parametrix, Inc, Bellevue, WA.

Weitkamp DE, Ruggerone GT. 2000. Factors affecting chinook populations: background report. Prepared for City of Seattle. Parametrix, Inc., Kirkland, WA.

West JE. 2001. Personal communication (data transmittal to Tad Deshler, Windward Environmental LLC, Seattle, WA). Washington Department of Fish and Wildlife, Marine Resources Division, Olympia, WA. March 14.

West JE, O'Neill SM, Lippert G, Quinnell S. 2001. Toxic contaminants in marine and anadromous fishes from Puget Sound, Washington. Results of the Puget Sound ambient monitoring program fish component 1989-1999. Washington Department of Fish and Wildlife, Olympia, WA.

Weston. 1993. Harbor Island remedial investigation report (part 2-sediment). Volume 1-report. Prepared for US Environmental Protection Agency, Region 10. Roy F. Weston, Inc., Seattle, WA.

Weston. 1999. Site inspection report, lower Duwamish River (RK 2.5-11.5), Seattle, Washington. Vol 1-Report and appendices. Prepared for US Environmental Protection Agency, Region 10. Roy F. Weston, Inc., Seattle, WA.

White MC, Chaney RL, Decker AM. 1979. Differential cultivar tolerance in soybean to phytotoxic levels of soil Zn. II. Range of Zn additions and the uptake and translocation of Zn, Mn, Fe, and P. Agronomy J 71:126-131.

WHO. 1993. Polychlorinated biphenyls and terphenyls. In: Dobson S, van Esch GJ, eds, Environmental health criteria 140, 2nd ed. World Health Organization, Geneva.

Widdows J, Page DS. 1993. Effects of tributyltin and dibutyltin on the physiological energetics of the mussel, *Mytilus edulis*. Marine Environ Res 35:233-249.

Williams MS. 1990. Terminal 107 (Kellogg Island), biological assessment 1989. Prepared for Port of Seattle. Parametrix , Bellevue ,WA.

Williams RW, Laramie RM, Ames JJ. 1975. A catalog of Washington streams and salmon utilization. Volume 1: Puget Sound region. Washington Department of Fisheries, Olympia, WA.

Windward. 2000. Results of second phase of clam reconnaissance survey. Memorandum to Doug Hotchkiss, Port of Seattle, July 19, 2000. Windward Environmental LLC, Seattle, WA.

Windward. 2001a. Problem formulation for Phase 1 ERA. Prepared for Lower Duwamish Waterway Group. Windward Environmental LLC, Seattle WA.

Windward. 2001b. Task 3: Study design for scoping-phase risk assessments: Conceptual model, exposure, and toxicity assessment for scoping-phase human health risk assessment. Prepared for US Environmental Protection Agency, Region 10, Seattle, WA and Washington State Department of Ecology, Northwest Field Office, Bellevue, WA. Windward Environmental LLC, Seattle, WA.

Windward. 2002. Lower Duwamish Waterway Remedial Investigation, Task 5: Identification of candidate sites for early actions - technical memorandum: Description of candidate site selection criteria. Prepared for Lower Duwamish Waterway Group for submittal to US Environmental Protection Agency, Seattle, WA and Washington Department of Ecology, Bellevue, WA. Windward Environmental LLC, Seattle, WA.

Wise MH, Linn LJ, Kennedy CR. 1981. A comparison of the feeding biology of mink (*Mustela vison*) and otter (*Lutra lutra*). J Zool Lond 195:181-213.

Wisk JD, Cooper KR. 1990. Comparison of the toxicity of several polychlorinated dibenzo-p-dioxins and 2,3,7,8-tetrachlorodibenzofuran in embryos of the Japanese medaka (*Oryzias latipes*). Chemosphere 20:361-377.

Wobeser G. 1975. Acute toxicity of methyl mercury chloride and mercuric chloride for rainbow trout (*Salmo gairdneri*) fry and fingerlings. J Fish Res Board Can 32:2005-2013.

Wobeser G, Nielsen ND, Schiefer B. 1976. Mercury and mink I. and II. The use of mercury contaminated fish as a food for rank mink. Can J Comp Med 40:30-33.

Wong PTS, Silverberg BA, Chau YK, Hodson PV. 1978. Lead and the aquatic biota. In: Nriagu JO, ed, The biochemistry of lead in the environment. Part B. Biological effects. Elsevier/North-Holland Biomedical Press, New York, NY, pp 279-342.

Wren CD, Hunter DB, Leatherland JF, Stokes PM. 1987. The effects of polychlorinated biphenyls and methylmercury, singly and in combination on mink. II: Reproduction and kit development. Arch Environ Contam Toxicol 16:449-454.

Wydoski RS, Whitney RR. 1979. Inland fishes of Washington. University of Washington Press, Seattle, WA.

Yates S. 1998. Marine wildlife from Puget Sound through the Inside Passage. Sasquatch Books, Seattle, WA.

Attachment A.1 GIS Maps: Published as Separate Document

Attachment A.2 Summary of King County Water Quality Assessment of Risks to Fish and Invertebrates in the Water Column

INTRODUCTION

This attachment provides select excerpts from and interpretation of the King County Water Quality Assessment (WQA) (King County 1999a,b,c,d) that assessed ecological and human health risks associated with chemicals and pathogens in the Duwamish River and Elliot Bay, and the extent of risk resulting from exposure to constituents associated with combined sewer overflows (CSOs). The risk assessment approach and results of this assessment underwent extensive review by a stakeholder committee and peer review panel, and were accepted by Washington Department of Ecology. This attachment summarizes the approach used to assess risks to fish and invertebrates associated with water column exposure under the baseline scenario (with CSOs), and presents the results obtained.

Risks to fish and invertebrates in the water column were evaluated in the WQA using a tiered approach based primarily on chemical concentrations in surface water. Risks to salmonids were evaluated separately through both water column exposures to chemicals and ingestion of PCBs, copper, and lead in their prey.[129] Concentrations of chemicals in surface water and sediment were based on the results of a detailed three-dimensional fate and transport hydrodynamic model calibrated with field data. The model divided the Duwamish Waterway between the downstream end of Harbor Island and the Turning Basin into 129 grid cells. Each grid cell is further divided into 10 water layers and one sediment layer, resulting in 1,290 water column and 129 sediment cells. The model predicted chemical concentrations in each grid element every 15 minutes for one year.

There were two types of model calibration performed. The first was a hydrodynamic calibration based on tides, velocity, salinity, etc. (see Section 4 of Appendix B1 of the King County WQA; King County 1999c). The second was a water quality calibration. The water quality calibration was performed with field data collected from 21 stations in the Duwamish River and Elliott Bay. Samples were collected over two 26-week periods from two depths (1 m below the surface and 1 m above the bottom) except for a few stations where water depths were too shallow to allow collection from two depths. Samples were also collected during storm conditions (for additional details on the field methods see Appendix A3 of the King County WQA; King County 1999b). For organic chemicals, such as PCBs and PAHs that were not detected in water samples, semipermeable membrane devices (SPMDs) and resident and transplanted mussels were used to estimate average ambient water column concentrations. Generally, the water quality model calibration entailed adjusting chemical load or mass inputs from sources until

[129] This summary does not present the King County salmonid ingestion (dietary) pathway results because that pathway was evaluated independently in the Phase 1 ERA.

simulated concentrations were comparable to corresponding observed field measurements (see Appendix B1, Section 5 of the King County WQAfor specific information on model calibration for metals and organic chemicals; King County 1999c).

METHODS

This section presents a summary of the methods used in the King County WQA. These include the screening of chemicals and the Tier 1 and 3 aquatic risk assessment methods.

Screening

Risks in the King County WQA were evaluated for chemicals of potential concern (COPCs) that had been identified for evaluation based on a screening process discussed in Appendix A.2, Section 4.5 of the WQA (King County 1999b). Briefly, chemicals were initially screened for their ability to cause human cancer. These chemicals were identified as COPCs. For non-cancer chemicals, each chemical's frequency of detection was reviewed. For chemicals detected greater than 5% of the time, the 95th percentile chemical concentrations[130] in water and sediment were compared against water quality criteria and sediment standards, respectively. Chemicals exceeding criteria or standards were identified as COPCs to be further evaluated in the risk assessment. Infrequently detected chemicals with method detection limits (MDLs) less than criteria/standards were not identified as COPCs and those with MDLs greater than criteria/standards were identified as posing uncertain risk. Based on this screening, 23 COPCs were identified for evaluation using a tiered approach, described below.

Tier 1

Tier 1 consisted of comparing exposure concentrations (developed through the fate and transport hydrodynamic model) to toxicity reference values (TRVs). For Tier 1, acute and chronic TRVs for each of the 23 COPCs were developed. Ten of these TRVs were based on marine Washington State water quality standards or marine federal water quality criteria (WQC). Seven TRVs for chemicals for which criteria have not been adopted, were based on toxicity studies from the literature, and where literature was lacking, six individual PAH TRVs were based on Quantitative Structure-Activity Relationships (QSARs). An uncertainty factor of 20, based on the approach in the Great Lakes Initiative (EPA 1995), was applied to the literature- and QSAR-based TRVs. TRVs were presented for both total recoverable and dissolved concentrations (see Appendix B4, Section 2.1 of the King County WQA for additional details; King County 1999a).

Surface water exposure concentrations were represented by the maximum monthly one-hour moving average and maximum monthly four-day moving average to represent reasonable worst-case acute and chronic exposures, respectively. For each chemical, acute and chronic TRVs (total recoverable for metals) were compared with the predicted worst-

[130] The 95th percentile on the sample population was calculated for water concentrations and the 95th percentile on the mean was calculated for sediment concentrations.

case acute and chronic total recoverable surface water concentrations[131] in every cell in the river for each month of the year. Ten COPCs had worst case total concentrations exceeding acute or chronic TRVs in at least one of the cells, as shown in Table 1, and were further evaluated in a second tier of the risk assessment.

As part of Tier 1, modeled acute and chronic exposure concentrations in surface water were also compared to TRVs specific for salmonid species obtained from USEPA ambient water quality criteria documents (see Appendix B4, Section 2.2 of the King County WQA for additional TRV details; King County 1999a). This comparison was specifically made to ensure that salmonids were protected (versus a more general ambient water quality criterion comparison). Water column exposure concentrations were less than the TRVs for salmonid species for all COPCs evaluated, and therefore, these COPCs were determined to not pose a risk to salmonids from water exposure.

Table 1. Chemicals with maximum total concentrations exceeding TRVs following the Tier 1 analysis.

COPC	Number of Months in the Year in which TRV was Exceeded	
	Acute TRV	Chronic TRV
Arsenic	2	0
Copper	12	12
Lead	1	5
Nickel	4	4
Zinc	11	0
TBT	0	2[a]
Total PCBs	0	5[a]
Benz(a)anthracene	1	0
Benzo(g,h,l)perylene	0	2
Fluoranthene	1	0

[a] Only 10 months were modeled for these COPCs because of initial model conditions.

Tier 3

The second tier was identified as Tier 3 in keeping with Water Environment Research Foundation methodology for aquatic ecological risk assessment (WERF 1996). In Tier 3, risk associated with surface water exposure to dissolved COCs (rather than total recoverable)[132] was evaluated based on the probability of affecting a given percentage of species, when sufficient toxicity data (based on dissolved concentrations) were available to develop a probability distribution for a particular COC. The first step of Tier 3 consisted of a compilation of genus mean acute and estimated chronic toxicity values for a range of organisms for each COC. A logistic regression model was then used to fit the

[131] Using total recoverable concentrations was considered a conservative method as not all of the metal is bioavailable (Protho 1993).

[132] Dissolved metal concentrations were used in the Tier 3 evaluations because they more closely represent the bioavailable fraction (Protho 1993).

toxicity data for each COC, and therefore, predict the percent of taxa affected at a given concentration (see Appendix B4, Section 2.4 of the King County WQA for additional details; King County 1999a). Risk characterization consisted of predicting the percent of species affected each month based on modeled maximum one-hour moving average and four-day moving average dissolved COC concentrations in each model cell and layer (i.e., every model cell was evaluated and no averaging across cells/grids occurred).

RESULTS

This section presents a summary of the King County WQA risk results for metals, TBT, PCBs and PAHs. Details of these results can be found in Appendix B4 of the King County WQA (King County 1999a).

Metals and TBT

Results of the logistic regression models for arsenic, copper, lead, nickel, zinc and TBT are presented in attached Figures 5-1 through 5-6 from Appendix B4 of the WQA.[133] These figures show the toxicity curves for each COC and the estimated exposure concentrations (EECs) in the Duwamish River.[134] The toxicity curves graphically show the distribution of toxicity values for a particular COC (see attached Figures 5-1 through 5-6 of the King County WQA), while the EECs are probability distributions of the modeled exposure concentrations. As shown in these figures, the acute and chronic EECs are generally substantially lower than concentrations predicted to affect aquatic organisms. Tier 3 risk predictions for metals were lower than Tier 1 (based on an HQ approach) because of the differences in dissolved versus total recoverable metals concentrations. Estimated acute and chronic risks in the LDW, expressed as percent of species affected, were less than or equal to one for all COCs except TBT and copper (see attached Summary Table 4-3 from Volume 1 of the WQA; King County 1999d).

Maximum monthly TBT chronic risks were estimated to only affect 2-4% of aquatic species, and those effects were only predicted in the vicinity of Harbor Island.No acute risks were identified for TBT (see Volume 1, Figure 4-5 of the King County WQA; King County 1999d). Maximum monthly chronic and acute copper risks from dissolved concentrations were predicted to affect 2-4% of aquatic species in a few select locations and less than or equal to 2% in all other locations (see Volume 1, Figures 4-3 and 4-4 of the King County WQA). Based on a USEPA recommended level of protection of at least 95% of species to ensure overall community function (Stephan et al. 1985, as cited in the King County WQA), these results indicate low risk to the aquatic community from exposure to surface water concentrations of COCs (i.e., 96% of the species are not

---

[133] Dissolved concentration data for lead and zinc had QA issues (i.e., most samples were flagged "B" for blank contamination). Dissolved data for lead and zinc were blank corrected prior to use in the model (see Section 2.1 and 3.3 in Appendix B1 of the King County WQA) so Tier 3 EECs should be representative of dissolved concentrations.

[134] Although risk estimates were determined for each model cell, the King County WQA only presents the results for the LDW as a whole. Specific results for each model cell were shown for copper and TBT, the COCs with the highest risk predictions, in Volume 1; Figures 4-3 through 4.5 of the King County WQA.

expected to be effected by copper or TBT). In this case, the most sensitive species, based on the probability curves, were bivalves (salmonid species were not as sensitive and were higher on the curve). The available water column field data also support these conclusions because no TRVs were exceeded based on maximum dissolved metals concentrations (see Table 2) or TBT water column concentrations estimated from wild and transplanted mussels[135] (see Table 3).

Table 2. Comparison of maximum measured metal concentrations in the Duwamish River (including East and West Waterway) to chronic TRVs

Metal	N	Max (µg/L)	Chronic TRV (µg/L)	HQ
Arsenic, dissolved	286	1.460	36	0.04
Cadmium, dissolved	287	0.083	8	0.01
Copper, dissolved	269	2.350	2.9	0.81
Lead, total	681	8.040	8.5	0.95
Mercury, dissolved	30	0.0007	1.1	0.001
Nickel, dissolved	263	1.500	7.9	0.19
Zinc, total	691	8.340	86	0.10

Total lead and zinc are shown because the dissolved data could not be used because of QA issues (i.e., most samples were flagged "B" for blank contamination). Total concentrations of lead and zinc were higher than dissolved concentrations, so HQs are higher than if dissolved concentrations had been used.

Chronic TRVs based on WA state water quality standards or EPA WQC (see Table 2-3 of Appendix B4, King County WQA). See Table 4-7 in the RI for water data.

Table 3. Estimated TBT water column concentrations and HQs based on mussel tissue samples at various locations in the Duwamish River

Sample Location	TBT (ng/L)			Chronic HQs		
	Resident	Trans- 1 m	Trans-3 m	Resident	Trans- 1 m	Trans-3 m
Slip4-wet	0.03			0.003		
Brandon-dry		0.03	0.06		0.003	0.006
Brandon-dry	0.02			0.002		
Brandon-wet		0.02	0.06		0.002	0.006
North of Slip #1-dry		0.03			0.003	
Kellogg-dry	0.03		0.03	0.003		0.003
Kellogg-wet		0.02	0.06		0.002	0.006
Term107-wet	0.01			0.001		
Duw/Dia-dry	0.04	0.04	0.03	0.004	0.004	0.003
Duw/Dia-wet	0.02			0.002		
Duw/Dia-wet		0.03	0.12		0.003	0.012
Average	0.02	0.03	0.06	0.0024	0.0028	0.006

[135] TBT was not measured in surface water samples. Resident and transplanted mussel samples were used to estimate TBT water concentrations using a bioconcentration factor of 3000 (see Section 5.3 of Appendix B1, King County WQA).

Sample Location	TBT (ng/L)			Chronic HQs		
	Resident	Trans- 1 m	Trans-3 m	Resident	Trans- 1 m	Trans-3 m
Maximum	0.04	0.04	0.12	0.004	0.004	0.012
Minimum	0.01	0.02	0.03	0.001	0.002	0.003

Resident: resident mussel samples

Trans 1 m: transplanted mussels 1 m below mean lower low water (MLLW)

Trans 3 m: transplanted mussels 3 m below MLLW

Wet: wet season sampling period

Dry: dry season sampling period

HQ= TBT water concentration (ng/L) divided by chronic TRV of 10 ng/L (EPA 1997).

PAHs and PCBs

The Tier 3 evaluation for three PAH compounds or for total PCBs could not be conducted because of insufficient toxicity data to develop a probability distribution. However, results for the Tier 1 analysis can be used to evaluate risk from water column PAH and PCB exposure.

Most of the PAH compounds evaluated had acute and chronic HQs less than 1.0 indicating low risk. However, benzo(a) anthracene and fluoranthene had maximum acute HQs of 1.4 and 1.1, respectively. Acute HQs only exceeded 1.0 for each PAH compound in one month of the year and in 0.08% and 0.4% of the cells evaluated (i.e., only 1 to 5 of the 1290 cells evaluated had HQs slightly greater than 1.0). For all other months of the year, acute HQs were below 1.0 and chronic HQs were always below 1.0. For benzo(g,h,i)perylene, the maximum chronic HQ was 1.4. In two of the modeled months, chronic HQs exceeded 1.0 but in only 0.2% to 3.6% of the cells evaluated (i.e., only 3 to 46 of the 1290 cells evaluated had HQs slightly greater than 1.0). In addition, if the field data for PAHs[136] were compared directly to their respective chronic TRVs, HQs would all be well below 1.0 (see Table 4). Therefore, overall aquatic life risks from water column PAH exposures were low (see Appendix B4, Tables 5-4 to 5-6 of the King County WQA for HQ summary statistics; King County 1999a).

[136] PAHs were not detected in surface water samples so semipermeable membrane devices (SPMDs) were used to estimate PAH surface water concentrations. SPMDs were deployed at two locations in the Duwamish River from March 26 to April 8, 1997. The 13-day deployment was considered sufficient for the analytes of interest to reach equilibrium between the SPMDs and the river water. Three SPMDs, including one field duplicate were deployed just offshore of the Duwamish/Diagonal combined sewer (CSO) outfall and two SPMDs were deployed just offshore of the Brandon Street CSO. Both sets of SPMDs were deployed in approximately 5 m of water (referenced to MLLW) at depths of 11 m and 3 m below the surface. SPMD samples were submitted to Battelle Marine Sciences Laboratory in Sequim, Washington for analysis.

Table 4. Estimated PAH water column concentrations and HQs based on SPMDs in the Duwamish River near Duwamish/Diagonal and Brandon CSOs

Compound	Duwamish/Diagonal		Brandon		Chronic TRV	Duwamish/Diagonal		Brandon	
	1 m (ng/L)	3 m (ng/L)	1 m (ng/L)	3 m (ng/L)		1 m (HQ)	3 m (HQ)	1 m (HQ)	3 m (HQ)
Phenanthrene	24.735	12.873	13.378	11.107	4,600	0.005	0.003	0.003	0.002
Fluoranthene	19.568	6.956	7.495	6.276	800	0.024	0.009	0.009	0.008
Pyrene	6.985	3.765	3.848	3.101	2,100	0.003	0.002	0.002	0.001
Benzo(a)anthracene	0.360	0.207	0.215	0.167	110	0.003	0.002	0.002	0.002
Chrysene	0.397	0.237	0.206	0.166	11,000	0.00004	0.00002	0.00002	0.00002
Benzo(b)fluoranthene	0.149	0.109	0.095	0.081	200	0.0007	0.0005	0.0005	0.0004
Benzo(k)fluoranthene	0.121	0.027	0.078	0.066	200	0.0006	0.0001	0.0004	0.0003
Benzo(a)pyrene	0.033	0.030	0.024	0.020	11,000	0.000003	0.000003	0.000002	0.000002
Indeno(1,2,3-c,d)pyrene	0.022	0.019	0.015	0.009	50	0.0004	0.0004	0.0003	0.0002
Dibenzo(a,h)anthracene	0.004	0.005	0.003	0.003	200	0.00002	0.00003	0.00002	0.00001
Benzo(g,h,i)perylene	0.048	0.061	0.030	0.027	50	0.0010	0.0012	0.0006	0.0005

See Table 2-3 of Appendix B4, King County WQA for source of chronic TRVs.

For PCBs, no acute HQs exceeded 1.0 in the Tier 1 assessment (maximum acute HQs ranged from 0.0006 to 0.1). Maximum chronic HQs exceeded 1.0 in five of ten months evaluated, and in 2.0% to 53.7% of the cells evaluated. The maximum chronic HQs ranged from 0.1 to 19 for all months evaluated, while the monthly average HQs ranged from 0.03 to 1.6, and median HQs ranged from 0.002 to 1.2 (see Appendix B4, Table 5-3 of the King County WQA for HQ summary statistics). Based on model calibration runs for organics, especially PCBs and TBT, the model tends to significantly over predict water column PCB concentrations (see Appendix B1, Section 5.4 of the King County WQA; King County 1999c). Calibration of the organic compounds was accomplished with many fewer samples than the metals, and thus has less certainty (e.g., PCBs were not detected in any water samples so SPMDs provided the only means to estimate water concentrations).

The chronic PCB TRV of 0.0049 µg/L was derived by applying a safety factor of 20 to the lowest chronic saltwater toxicity value (0.098 µg/L) contained in the EPA criteria document (EPA 1980). Without the use of the safety factor, no chronic HQs would have exceeded 1.0. In addition, if the field data for PCBs[137] were compared directly to the chronic PCB TRV, HQs would all be below 1.0 (see Table 5).

[137] PCBs were not detected in surface water samples so SPMDs were used to estimate PCB surface water concentrations. Samples were analyzed for both PCB Aroclors and PCB congeners.

Table 5. Estimated PCB water column concentrations and HQs based on SPMDs in the Duwamish River near Duwamish/Diagonal and Brandon CSOs

Compound	Units	Duwamish/Diagonal		Brandon	
		1 m	3 m	1 m	3 m
PCB8	ng/L	0.000	0.000	0.000	0.000
PCB18	ng/L	0.041	0.019	0.022	0.015
PCB28	ng/L	0.021	0.015	0.011	0.008
PCB52	ng/L	0.031	0.017	0.020	0.017
PCB49	ng/L	0.049	0.038	0.032	0.027
PCB44	ng/L	0.016	0.009	0.010	0.009
PCB66	ng/L	0.000	0.000	0.000	0.000
PCB101	ng/L	0.013	0.007	0.008	0.006
PCB87	ng/L	0.003	0.002	0.002	0.002
PCB77	ng/L	0.000	0.000	0.000	0.000
PCB118	ng/L	0.004	0.002	0.003	0.002
PCB153	ng/L	0.004	0.002	0.002	0.002
PCB105	ng/L	0.003	0.002	0.002	0.001
PCB138	ng/L	0.003	0.002	0.002	0.001
PCB187	ng/L	0.001	0.0004	0.0004	0.0002
PCB128	ng/L	0.0005	0.001	0.0005	0.000
PCB180	ng/L	0.001	0.0002	0.001	0.001
PCB170	ng/L	0.002	0.000	0.000	0.000
PCB195	ng/L	0.000	0.00001	0.000	0.000
Total PCBs[a] (congener based)	ng/L	0.195	0.117	0.117	0.092
Total PCB HQ[b] (congener based)	unitless	0.04	0.02	0.02	0.02
Aroclor 1242 (max)[c,d]	ng/L	1.244	0.682	0.892	0.677
Aroclor 1242 (avg)[d,e]	ng/L	0.150	0.082	0.107	0.081
Aroclor 1254 (max)[c,d]	ng/L	1.375	0.813	0.819	0.667
Aroclor 1254 (avg)[d,e]	ng/L	0.165	0.098	0.098	0.080
1242 + 1254 (max)[c,e]	ng/L	2.619	1.496	1.711	1.344
1242 + 1254 (avg)[d,e]	ng/L	0.315	0.180	0.206	0.162
1242 + 1254 (max) HQ [b]	Unitless	0.53	0.31	0.35	0.27
1242 + 1254 (avg) HQ [b]	Unitless	0.06	0.04	0.04	0.03

[a] Total PCBs concentrations based on the above congeners likely underestimate actual total PCB concentrations because not all congeners were analyzed due to lack of congener-specific partition coefficients.

[b] HQ=Hazard quotients based on a 4.9 ng/L chronic PCB TRV.

[c] Max Aroclor concentration estimated based on maximum partition coefficient developed by Battelle for the congeners listed above.

[d] Aroclor concentrations estimated because a partition coefficient could not be developed for either Aroclor mixture. Only detected Aroclor mixtures shown.

[e] Average Aroclor concentration estimated based on average of partition coefficients developed by Battelle for the congeners listed above.

These results indicate that there were negligible acute (short-term) risks from PCB exposure in water, and the chronic risks to aquatic organisms are were also negligible based on water exposures measured and modeled in the LDW.

CONCLUSION

An analysis of risks to water column invertebrates and fish suggests that there is low to negligible risk associated with predicted water column concentrations based on a model calibrated with field data. This assessment was conducted to assess risks to species from water concentrations resulting from all current chemical sources in the LDW.

REFERENCES FOR ATTACHMENT A-2

Anchor Environmental. 2001. Duwamish/Diagonal CSO/SD Alternatives Evaluation Report, Report prepared for Elliott Bay/Duwamish Restoration Program Panel by Anchor Environmental, LLC, King County Department of Natural Resources, and EcoChem, Inc. October 2001.

EVS Consultants and Hart Crowser. 1995. Supplementary Remedial Investigation Chemical Data Summary Report, Harbor Island Sediment Operable Unit, prepared for the Harbor Island Sediment Work Group c/o Port of Seattle, July 1995.

EPA. 1980. Ambient water quality criteria for polychlorinated biphenyls. Report 440/5-80-068. US Environmental Protection Agency, Washington, DC.

EPA. 1995. Final water quality guidance for the Great Lakes System: Final rule. 40 CFR 9, 122, 123, 131, and 132.

EPA. 1997. Ambient aquatic life water quality criteria for tributyltin-1997. Office of Water, Health and Ecological Criteria Division. US EPA , Washington, D.C. EPA-822-D-97-001.

GeoSea Consulting, Ltd. 1994. Sediment Transport in Elliott Bay and the Duwamish River, Seattle: Implications To Estuarine Management. Prepared for Washington Department of Ecology, Toxics Cleanup Program.

King County. 1999a. King County combined sewer overflow water quality assessment for the Duwamish River and Elliott Bay. Appendix B2, B3, & B4: Human health, wildlife, and aquatic life risk assessments. King County Department of Natural Resources. Seattle, WA.

King County. 1999b. King County combined sewer overflow water quality assessment for the Duwamish River and Elliott Bay. Appendix A1, A2, A3: Problem Formulation, Analysis Plan, and Field Sampling Workplan. King County Department of Natural Resources. Seattle, WA.

King County. 1999c. King County combined sewer overflow water quality assessment for the Duwamish River and Elliott Bay. Appendix B1: Hydrodyamnic and Fate and Transport Numerical Model. King County Department of Natural Resources. Seattle, WA.

King County. 1999d. King County combined sewer overflow water quality assessment for the Duwamish River and Elliott Bay. Volume 1: Overview and Interpretation Report. King County Department of Natural Resources. Seattle, WA.

King County. 2001. King County combined sewer overflow water quality assessment for the Duwamish River and Elliott Bay. Working draft Appendix E. King County Department of Natural Resources. Seattle, WA.

Patmont, C.R., M.E. Harper, and others, 1983. Water Quality Assessment of the Duwamish Estuary, Washington, prepared for the Municipality of Metropolitan Seattle, May 1983.

Prothro MG. 1993. Office of Water policy and technical guidance on interpretation and implementation of aquatic life metals criteria. U.S. EPA, Office of Water. Washington, D.C. 7 pp + appendices.

Stephan CE, Mount DI, Hansen DJ, Gentile JH, Chapman GA, Brungs WA. 1985. Guidelines for deriving numerical national water quality criteria for the protection of aquatic organisms and their uses. PB85-227094. U.S. Environmental Protection Agency, Office of Research and Development, Washington, DC. 98 pp.

Water Environment Research Foundation (WERF). 1996. Methodology for aquatic ecological risk assessment. Project No. RP91-AER-1.

Figures and tables from King County WQA

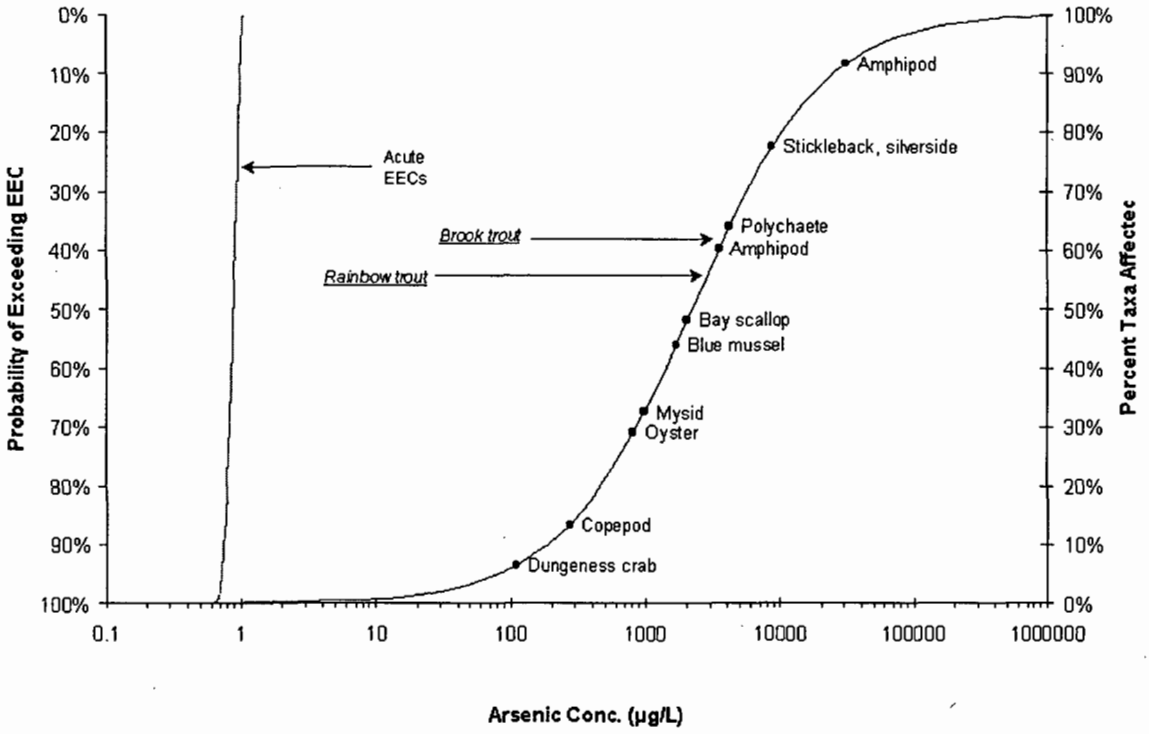

Figure 5-1. Acute EEC and Marine Toxicity Distributions for Dissolved Arsenic

Figure 5-1 of King County WQA

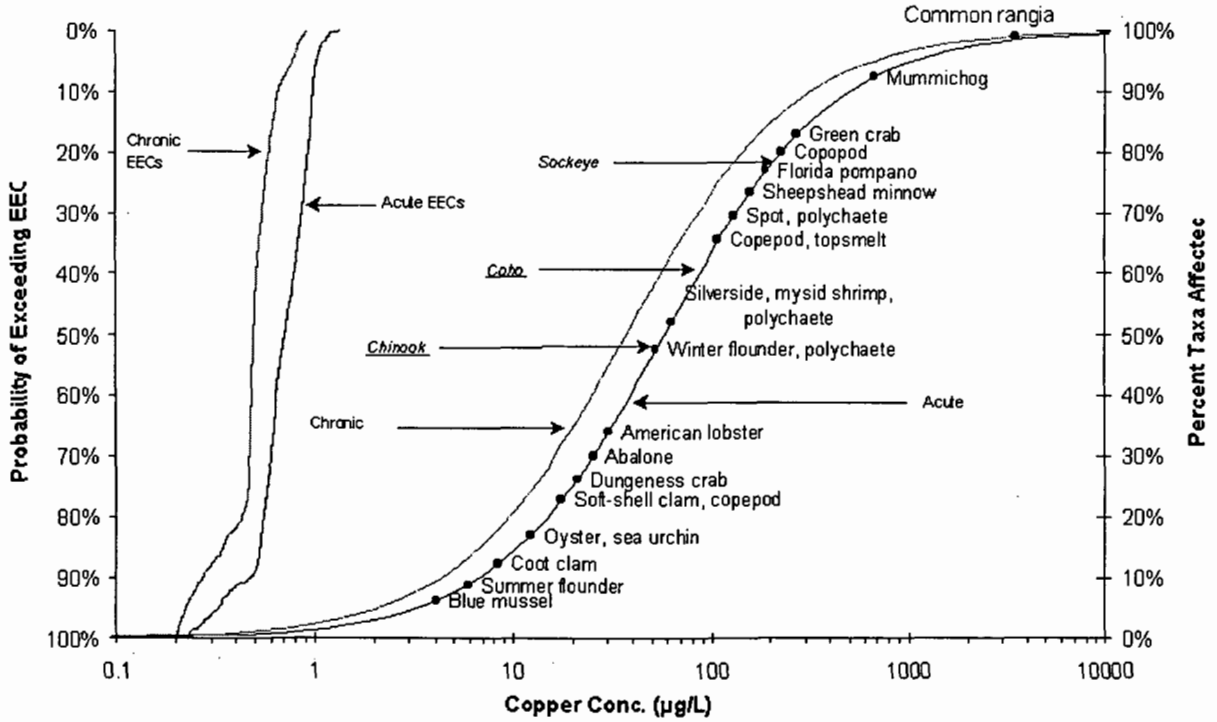

Figure 5-2. Acute and Chronic EEC and Marine Toxicity Distribution for Dissolved Copper

Figure 5-2 of King County WQA

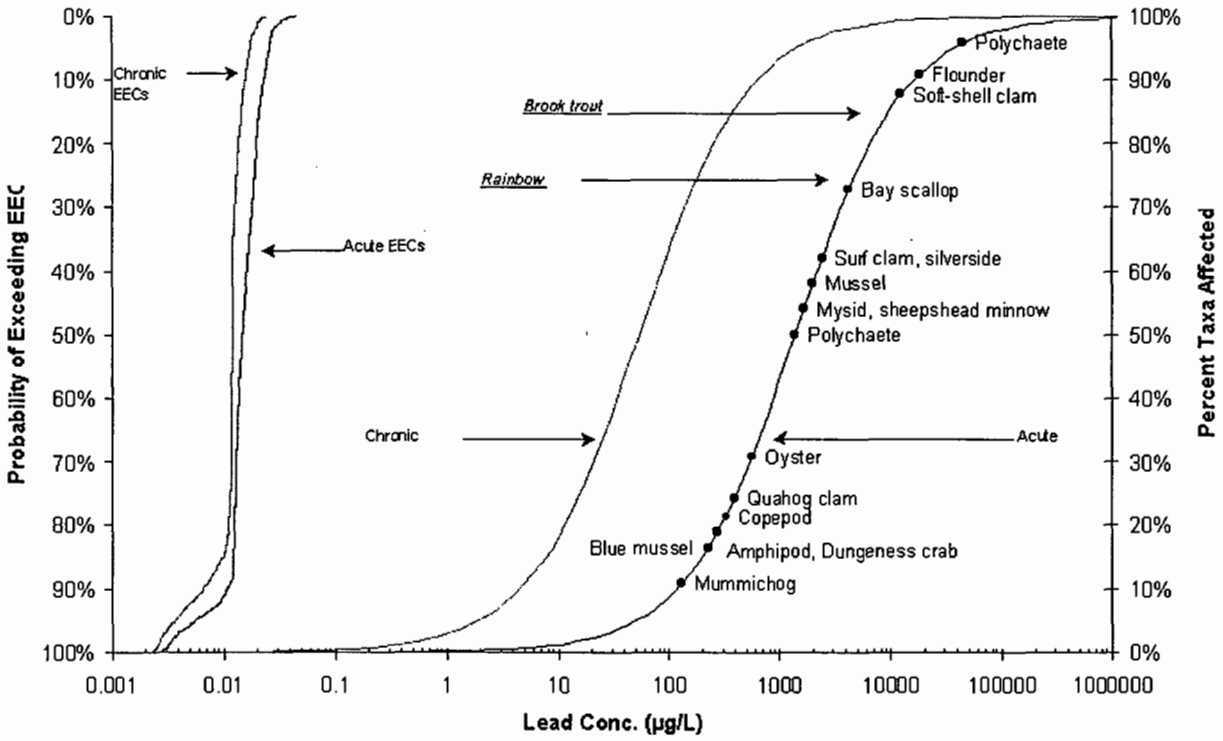

Figure 5-3. Acute and Chronic EEC and Marine Toxicity Distribution for Dissolved Lead

Figure 5-3 of King County WQA

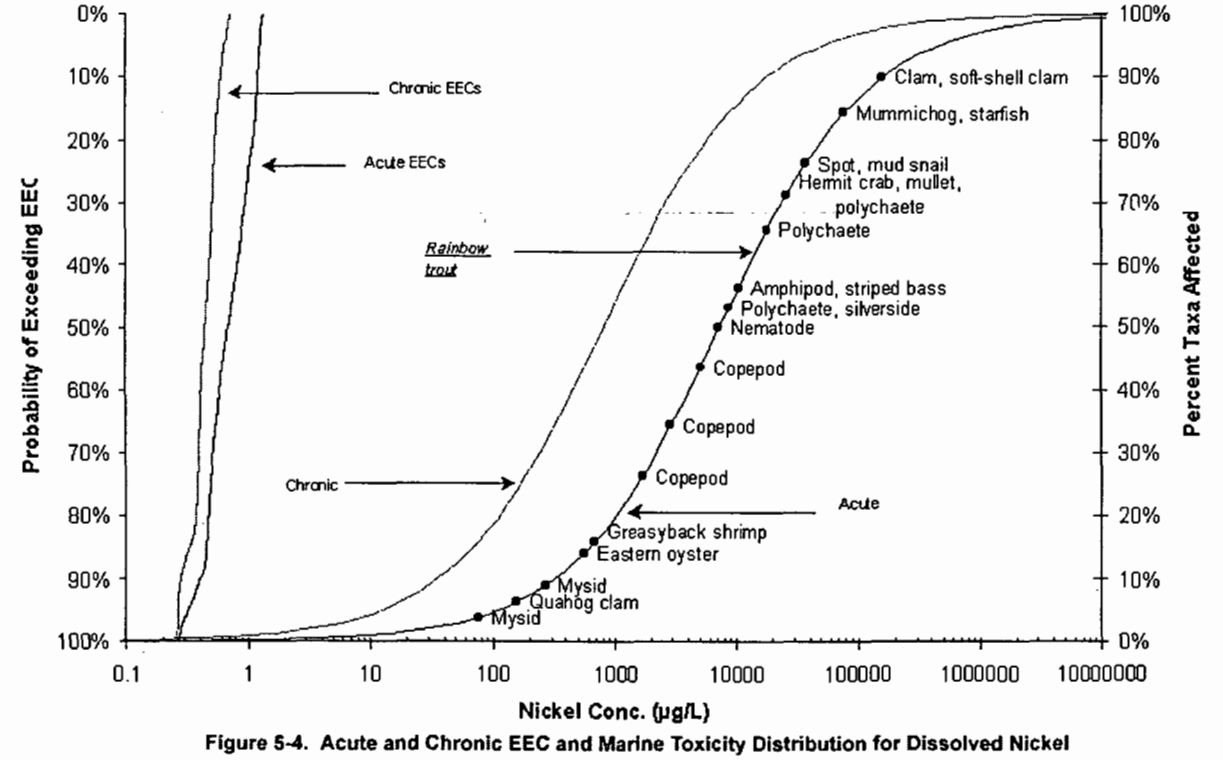

Figure 5-4. Acute and Chronic EEC and Marine Toxicity Distribution for Dissolved Nickel

Figure 5-4 of King County WQA

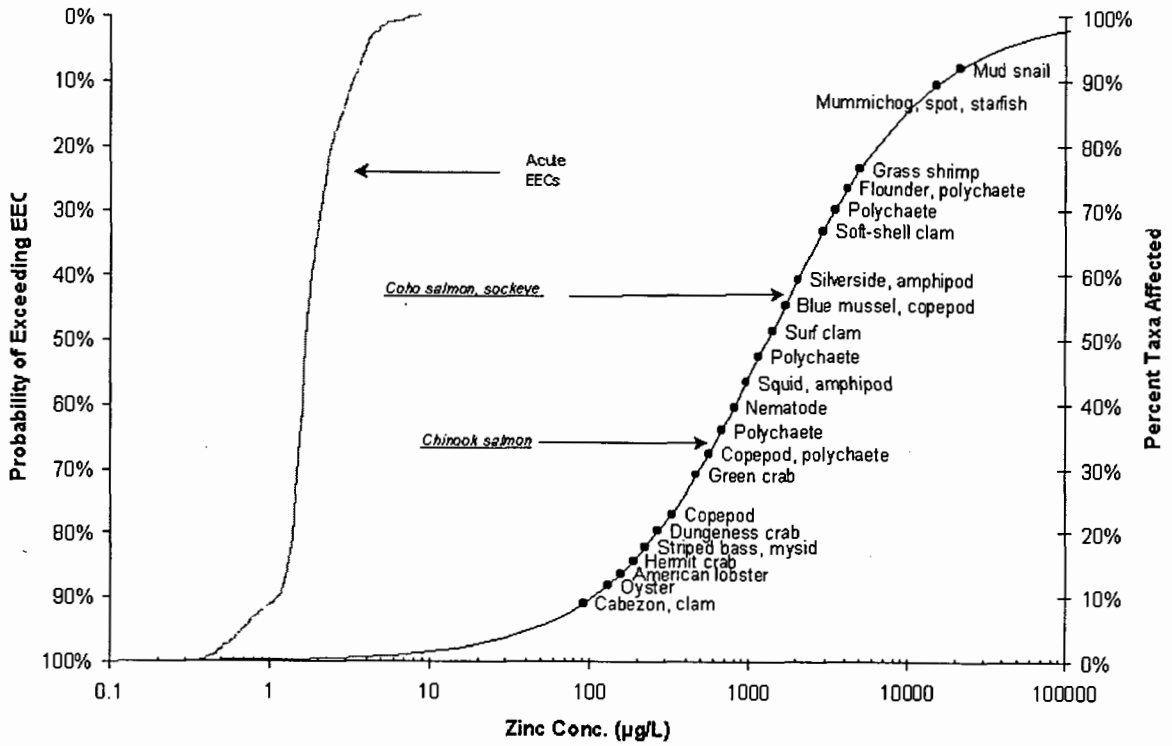

Figure 5-5. Acute EEC and Marine Toxicity Distribution for Dissolved Zinc

Figure 5-5 of King County WQA

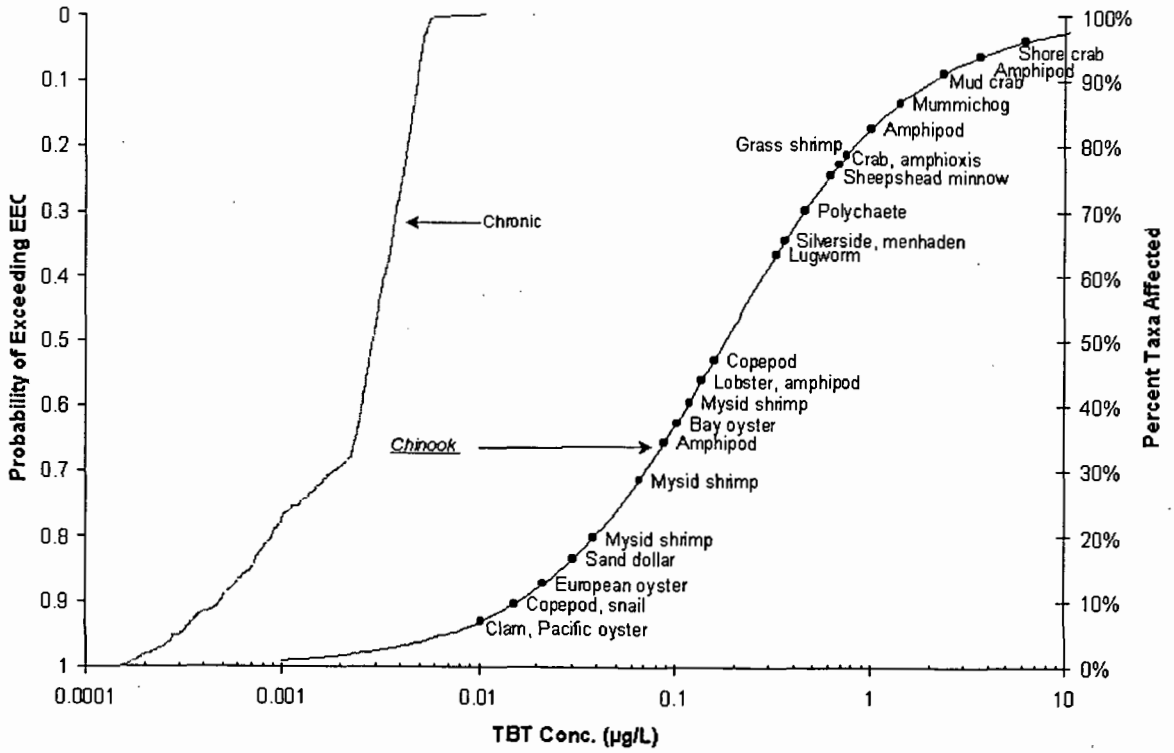

Figure 5-6. Chronic EEC and Marine Toxicity Distribution for Dissolved TBT

Figure 5-6 of King County WQA

Table 4-3. [from Volume 1 of King County WQA] Percent Aquatic Life Species at Acute and Chronic Risk from Exposure to COPCs[a] in the Study Area (Values Presented are Baseline, Without CSOs)

Chemical	Sep	Oct	Nov	Dec	Jan	Feb	Mar	Apr	May	Jun	Jul	Aug
Duwamish River, Acute Risks												
Arsenic	<1%,<1%	<1%,<1%	<1%,<1%	<1%,<1%	<1%,<1%	<1%,<1%	<1%,<1%	<1%,<1%	<1%,<1%	<1%,<1%	<1%,<1%	<1%,<1%
Copper	1%,1%	1%,1%	1%,1%	1%,1%	1%,1%	1%,1%	1%,1%	1%,1%	1%,1%	1%,1%	1%,1%	1%,1%
Lead	<1%,<1%	<1%,<1%	<1%,<1%	<1%,<1%	<1%,<1%	<1%,<1%	<1%,<1%	<1%,<1%	<1%,<1%	<1%,<1%	<1%,<1%	<1%,<1%
Nickel	<1%,<1%	<1%,<1%	<1%,<1%	<1%,<1%	<1%,<1%	<1%,<1%	<1%,<1%	<1%,<1%	<1%,<1%	<1%,<1%	<1%,<1%	<1%,<1%
Zinc	<1%,<1%	<1%,<1%	<1%,<1%	<1%,<1%	<1%,<1%	<1%,<1%	<1%,<1%	<1%,<1%	<1%,<1%	<1%,<1%	<1%,<1%	<1%,<1%
Duwamish River, Chronic Risks												
Copper	1%,1%	2%,2%	1%,1%	1%,1%	1%,1%	1%,1%	1%,1%	1%,1%	1%,1%	1%,1%	1%,1%	1%,1%
Lead	<1%,<1%	<1%,<1%	<1%,<1%	<1%,<1%	<1%,<1%	<1%,<1%	<1%,<1%	<1%,<1%	<1%,<1%	<1%,<1%	<1%,<1%	<1%,<1%
Nickel	1%,1%	1%,1%	<1%,<1%	<1%,<1%	<1%,<1%	<1%,<1%	1%,1%	<1%,<1%	<1%,<1%	<1%,<1%	<1%,<1%	<1%,<1%
TBT	N/AV[b]	N/AV[b]	2%,2%	1%,1%	1%,1%	<1%,<1%	1%,2%	<1%,<1%	<1%,<1%	<1%,<1%	<1%,<1%	<1%,<1%
Elliott Bay, Acute Risks												
Arsenic	<1%,<1%	<1%,<1%	<1%,<1%	<1%,<1%	<1%,<1%	<1%,<1%	1%,1%	<1%,<1%	<1%,<1%	<1%,<1%	<1%,<1%	<1%,<1%
Copper	1%,1%	<1%,<1%	<1%,<1%	<1%,<1%	<1%,<1%	<1%,<1%	1%,<1%	<1%,<1%	1%,1%	<1%,<1%	<1%,<1%	<1%,<1%
Elliott Bay, Chronic Risks												
Copper	1%,1%	1%,1%	1%,1%	<1%,<1%	<1%,<1%	1%,1%	1%,1%	1%,1%	1%,1%	1%,1%	1%,1%	1%,1%
Lead	<1%,<1%	<1%,<1%	<1%,<1%	<1%,<1%	<1%,<1%	<1%,<1%	<1%,<1%	<1%,<1%	<1%,<1%	<1%,<1%	<1%,<1%	<1%,<1%
Nickel	<1%,<1%	<1%,<1%	<1%,<1%	<1%,<1%	<1%,<1%	<1%,<1%	3%,3%	<1%,<1%	<1%,<1%	<1%,<1%	<1%,<1%	<1%,<1%
TBT	2%,2%	<1%,<1%	<1%,<1%	<1%,<1%	<1%,<1%	<1%,<1%	<1%,1%	<1%,<1%	<1%,<1%	<1%,<1%	<1%,<1%	<1%,<1%

[a] Only chemicals that exceeded their screening thresholds are presented here.

[b] N/AV - Not available due to model irregularities

Attachment A.3. Wildlife Tables from King County WQA

Note: Body weights and ingestion rates were not available from King County to document the derivation of LOAELs and NOAELs in those few cases where doses were derived from dietary concentrations presented in the literature.

Table 2-2. TRVs for the River Otter

Analyte	Literature LOAEL (mg/kg/d)	Scaled LOAEL (mg/kg/d)	Literature NOAEL (mg/kg/d)	Scaled NOAEL (mg/kg/d)	Test Organism	Effect	References
Metals/Metalloids							
Arsenic	1.26	0.52	0.126[a]	0.052	Rat	Decreased litter size	ATSDR (1991d); Schroder and Mitchener (1971)
Cadmium	1.9	0.4	1	0.2	Mouse	Reproductive failure	ATSDR (1991b); Schroeder and Mitchener (1971); ORNRL (1996)
Copper	15.1	10.1	11.7	7.8	Mink	Kit mortality	ORNRL (1996); Aulerich et al. (1982)
Lead	1.5	0.3	0.15[a]	0.04	Mouse	Reproductive success of implanted ova	Eisler (1988); Clark, (1979)
Mercury (inorganic)	3	1.2	0.09	0.06	Rat, mink	Kidney damage (rat), no clinical/ pathological signs of tox. (mink)	Carmignani et al. (1989); Wobeser et al. (1976)
Nickel	80	33	40	17	Rat	Decreased offspring per litter	ORNRL (1996); Ambrose et al. (1976)
Zinc	320	132	160	66	Rat	Increased fetal resorption	Schlicker and Cox (1968)
Organometallics							
Tributyltin	3.4	1.4	0.34[a]	0.14	Rat	Decreased pup weight	IRIS (1998)
Polychlorinated Biphenyls							
Aroclor 1016	3.43	2.29	1.37	0.91	Mink	Reproductive effects	Eisler (1986); Ringer (1983)
Aroclor 1221	3.43	2.29	0.447	0.298	Mink	Reproductive effects	Eisler (1986); Ringer (1983)

Table 2-2. TRVs for the River Otter (continued)

Analyte	Literature LOAEL (mg/kg/d)	Scaled LOAEL (mg/kg/d)	Literature NOAEL (mg/kg/d)	Scaled NOAEL (mg/kg/d)	Test Organism	Effect	References
Aroclor 1232	0.34	0.23	0.14	0.09	Mink	Fertility, whelping, number of kits	Wren et al. (1987); Hornshaw et al. (1983)
Aroclor 1242	1.12	0.75	0.447	0.298	Mink	Reproductive failure	Eisler (1986); Ringer (1983)
Aroclor 1248	0.34	0.23	0.14	0.09	Mink	Fertility, whelping, number of kits	Keplinger et al. (1971)
Aroclor 1254	0.34	0.23	0.14	0.09	Mink	Fertility, whelping, number of kits	Wren et al. (1987); Hornshaw et al. (1983)
Aroclor 1260	6	2.48	0.06	0.03	Rat	Stillborns and pup survival	NAS (1979); Burke and Fitzhugh (1970); Keplinger et al. (1971).
Total PCBs	0.34	0.23	0.14	0.09	Mink		
Organics							
1,4-Dichlorobenzene	600	139	40[a]	17	Mouse, rat	Liver degeneration, decreased white blood cell count (mouse), no effects on liver or immune system (rat)	ATSDR (1991a); Gaines and Linder (1986); NTP (1987)
4-Methylphenol	N/AV[b]	N/AV[b]	450	186	Rat	Reproduction	ATSDR (1990); BRRC (1989)
Benzo(a)anthracene[c]	10	2.3	1[a]	0.2	Mouse	Reproductive effects	MacKenzie & Angevine (1981)
Benzo(a)pyrene	10	2.3	1[a]	0.2	Mouse	Reproductive effects	MacKenzie & Angevine (1981)
Benzo(e)pyrene	10	2.3	1[a]	0.2	Mouse	Reproductive effects	MacKenzie & Angevine (1981)
Benzo(b)fluoranthene[c]	10	2.3	1[a]	0.2	Mouse	Reproductive effects	MacKenzie & Angevine (1981)

Table 2-2. TRVs for the River Otter (continued)

Analyte	Literature LOAEL (mg/kg/d)	Scaled LOAEL (mg/kg/d)	Literature NOAEL (mg/kg/d)	Scaled NOAEL (mg/kg/d)	Test Organism	Effect	References
Benzo(g,h,I)perylene	10	2.3	1[a]	0.2	Mouse	Reproductive effects	MacKenzie & Angevine (1981)
Benzo(k)fluoranthene[c]	10	2.3	1[a]	0.2	Mouse	Reproductive effects	MacKenzie & Angevine (1981)
Bis(2-Ethylhexyl) phthalate	183.3	42.56	18.3	4.25	Mouse	Reproductive effects	ORNRL 1996; Lamb et al. (1987)
Chrysene[c]	10	2.3	1[a]	0.2	Mouse	Reproductive effects	MacKenzie & Angevine (1981)
Dibenzo(a,h)anthracene[c]	10	2.3	1[a]	0.2	Mouse	Reproductive effects	MacKenzie & Angevine (1981)
Fluoranthene	250	58.1	125	29.0	Mouse	Systemic	IRIS (1998)
Indeno(1,2,3-cd)pyrene[b]	10	2.3	1[a]	0.2	Mouse	Reproductive effects	MacKenzie & Angevine (1981)
Pyrene	125	29.0	75	17	Mouse	Systemic	HEAST (1995)
Phenanthrene[b]	10	2.3	1[a]	0.2	Mouse	Reproductive effects	MacKenzie & Angevine (1981)

[a] The NOAEL was estimated from the LOAEL using an uncertainty factor of 10.
[b] N/AV = Not Available
[c] LOAEL and NOAEL estimated using benzo(a)pyrene as a "surrogate" PAH.

Table 2-3. TRVs for Avian Receptors

Analyte	Literature LOAEL (mg/kg/d)	Heron LOAEL[a] (mg/kg/d)	Eagle, Sandpiper LOAEL[b] (mg/kg/d)	Literature NOAEL (mg/kg/d)	Heron Level NOAEL[a] (mg/kg/d)	Eagle, Sandpiper NOAEL[b] (mg/kg/d)	Test Organism	Effect	References
Metals/Metalloids									
Arsenic	12.8	6.4	2.6	5.14	2.57	1.03	Mallard	Mortality	ORNRL (1996); USFWS (1964)
Cadmium	4.4	2.2	0.9	1.45	0.73	0.29	Chicken, mallard	Decreased egg production	NRC (1980); Leach et al. (1979); Scheuhammer (1987); White and Finley (1978)
Copper	61.7	30.9	12.3	47	24	9.4	Chicken	Weight gain and mortality	ORNRL (1996); Mehring et al. (1960)
Lead	0.72	0.36	0.14	0.072	0.036	0.014	Japanese quail	Delayed egg production	Scheuhammer (1987); Edens et al. (1976)
Mercury (inorganic)	0.74	0.37	0.15	0.37	0.19	0.07	Japanese quail	Eggshell thinning	Stoewsand et al. (1971)
Nickel	107	53.5	21.4	77	39	15	Mallard	Mortality and reduced growth	ORNRL (1996); Cain and Pafford (1981)
Zinc	137	68.5	27.4	131	65.5		Chicken	Reduced hatchability	Eisler (1993); Stahl et al. (1990)
Zinc				90		18	Chicken	Decreased growth (individual effect)	Roberson and Schaible (1960)

Table 2-3. TRVs for Avian Receptors (continued)

Analyte	Literature LOAEL (mg/kg/d)	Heron LOAEL[a] (mg/kg/d)	Eagle, Sandpiper LOAEL[b] (mg/kg/d)	Literature NOAEL (mg/kg/d)	Heron Level NOAEL[a] (mg/kg/d)	Eagle, Sandpiper NOAEL[b] (mg/kg/d)	Test Organism	Effect	References
Organometallics									
Tributyltin	16.9	8.45	3.38	6.8	3.4	1.36	Japanese quail	Reduced hatchability and egg weight	ORNRL (1996); Schlatter et al. (1993)
Polychlorinated Biphenyls									
Aroclor 1016	0.91	0.46	0.18	1.83	0.92	0.37	Chicken	Egg hatchability, teratogenic effects	Cecil et al. (1974)
Aroclor 1221	0.91	0.46	0.18	1.83	0.92	0.37	Chicken	Egg hatchability, teratogenic effects	Cecil et al. (1974)
Aroclor 1232	0.91	0.46	0.18	0.46	0.23	0.09	Chicken	Egg hatchability	Lillie et al. (1975)
Aroclor 1242	0.91	0.46	0.18	0.46	0.23	0.09	Chicken	Egg hatchability	Lillie et al. (1975)
Aroclor 1248	0.91	0.46	0.18	0.46	0.23	0.09	Chicken	Egg hatchability	Lillie et al. (1975)
Aroclor 1254	0.99	0.50	0.20	0.46	0.23	0.09	Ringed turtle dove, chicken	Egg hatchability	Heinz et al. (1984); Lillie et al. (1975); Hill et al. (1976); Scott (1977)
Aroclor 1260	0.91	0.46	0.18	0.46	0.23	0.09	Chicken	Egg hatchability	Lillie et al. (1975)
Total PCBs	0.91	0.46	0.18	0.46	0.23	0.09	Chicken	Egg hatchability	Lillie et al. (1975)

Table 2-3. TRVs for Avian Receptors (continued)

Analyte	Literature LOAEL (mg/kg/d)	Heron LOAEL[a] (mg/kg/d)	Eagle, Sandpiper LOAEL[b] (mg/kg/d)	Literature NOAEL (mg/kg/d)	Heron Level NOAEL[a] (mg/kg/d)	Eagle, Sandpiper NOAEL[b] (mg/kg/d)	Test Organism	Effect	References
Organics									
1,4-Dichlorobenzene	600	300	120	40	20	8.0	Rat, mouse	Liver degeneration, decreased white blood cell count (mouse), no effects on liver or immune system (rat)	ATSDR (1991a); Gaines and Linder (1986); NTP (1987); Carlson and Tardiff (1976).
4-Methylphenol	22.6[c]	11.3	4.5	9.42[c]	4.71	1.88	Red-winged blackbird	Mortality	RTECS (1995); Schaeffer et al. (1983)
Benzo(a)anthracene	10	5	2	1[d]	0.5	0.2	Mouse	Reproductive effects	MacKenzie & Angevine (1981)
Benzo(b)fluoranthene	10	5	2	1[d]	0.5	0.2	Mouse	Reproductive effects	MacKenzie & Angevine (1981)
Benzo(k)fluoranthene	10	5	2	1[d]	0.5	0.2	Mouse	Reproductive effects	MacKenzie & Angevine (1981)
Benzo(a)pyrene [a, c]	10	5	2	1[d]	0.5	0.2	Mouse	Reproductive effects	MacKenzie & Angevine (1981)
Benzo(e)pyrene	10	5	2	1[d]	0.5	0.2	Mouse	Reproductive effects	MacKenzie & Angevine (1981)
Benzo(g,h,i)perylene	10	5	2	1[d]	0.5	0.2	Mouse	Reproductive effects	MacKenzie & Angevine (1981)

Table 2-3. TRVs for Avian Receptors (continued)

Analyte	Literature LOAEL (mg/kg/d)	Heron LOAEL[a] (mg/kg/d)	Eagle, Sandpiper LOAEL[b] (mg/kg/d)	Literature NOAEL (mg/kg/d)	Heron Level NOAEL[a] (mg/kg/d)	Eagle, Sandpiper NOAEL[b] (mg/kg/d)	Test Organism	Effect	References
Bis(2-Ethylhexyl) phthalate	N/AV[e]	N/AV[e]	N/AV[e]	1.11	0.56	0.22	Ringed dove	Reproductive effects	ORNRL (1996); Peakall (1974)
Chrysene	10	5	2	1[d]	0.5	0.2	Mouse	Reproductive effects	MacKenzie & Angevine (1981)
Dibenzo(a,h) anthracene	10	5	2	1[d]	0.5	0.2	Mouse	Reproductive effects	MacKenzie & Angevine (1981)
Fluoranthene	250	125	50	125	63	25	Mallard	Reproductive effects	HEAST (1995)
Indeno (1,2,3-cd) pyrene	10	5	2	1[d]	0.5	0.2	Mouse	Reproductive effects	MacKenzie & Angevine (1981)
Phenanthrene	10	5	2	1[d]	0.5	0.2	Mouse	Reproductive effects	MacKenzie & Angevine (1981)
Pyrene	125	63	25	75	38	15	Mallard	Reproductive effects	HEAST (1995)

[a] The population level NOAEL or LOAEL is based on the NOAEL or LOAEL divided by an uncertainty factor of 2 to account for interspecies variability.

[b] The individual level NOAEL or LOAEL is based on the NOAEL or LOAEL divided by an uncertainty factor of 5 to account for potentially more sensitive endpoints such as systemic effects of growth.

[c] The LOAEL and NOAEL are based on an uncertainty factor of 5 and 12, respectively, for the ratio of acute and chronic effect doses for 3-methylphenol in rats (it was assumed the ration is the same for birds).

[d] The NOAEL was estimated from the LOAEL using an uncertainty factor of 10.

[e] N/AV = Not Available

Table 3-2. Great Blue Heron Body Weight (kg) Summary Statistics

Sex	Mean	SD	SE	n	Reference
Male	2.576	0.299	0.0725	17	EPA (1993f)
Female	2.204	0.337	0.0870	15	EPA (1993f)

Table 3-3. Bald Eagle Body Weight (kg) Summary Statistics

Sex	Mean	SD[a]	Range	n	Reference
Male	4.13	0.197	3.637-4.819	35	Dunning (1993); EPA (1993f)
Female	5.35	0.462	3.631-6.4	37	Dunning (1993); EPA (1993f)
Male	4.325	NA	NA	52	Stalmaster (1987)
Female	5.268	NA	NA	54	Stalmaster (1987)

[a] Standard deviation estimated as 1/6 the range of body weights

Table 3-4. Spotted Sandpiper Body Weight (kg) Summary Statistics

Sex	Mean	SD[a]	Range	n	Reference
Male	0.0379	0.0018	0.034-0.041	8	EPA (1993); Maxson and Oring (1980)
Female	0.0471	0.0018	0.043-0.050	9	EPA (1993); Maxson and Oring (1980)

[a] Standard deviation estimated as 1/4 the range of body weights

Table 3-5. River Otter Body Weight (kg) Summary Statistics

Sex	Mean	SE	n	Reference
Male	9.2	0.6	4	EPA (1993f); Melquist and Hornocker (1983)
Female	7.9	0.2	6	EPA (1993f); Melquist and Hornocker (1983)

Table 3-6. Water and Sediment EEC Summary Table for the Heron Patch

Table 3-6. Water and Sediment EEC Summary Table for the Heron Patch

Chemical	Water (µg/L)						Sediment (mg/kg dry weight)				
	With CSOs		Without CSOs		Reference Site		With CSOs		Without CSOs		Refe
	Mean	Standard Error	Mean	Standard Error	Mean	Standard Error	Mean	Standard Error	Mean	Standard Error	Mean
1,4-Dichlorobenzene	2.65E-03	1.35E-03	2.59E-03	1.36E-03	N/AV	N/AV	9.67E-02	2.27E-03	9.66E-02	2.28E-03	N/AV
4-Methylphenol	1.70E-02	2.42E-03	1.61E-02	2.47E-03	N/AV	N/AV	9.59E-02	5.63E-03	9.63E-02	5.72E-03	N/AV
Arsenic	1.34E+00	1.03E-01	1.34E+00	1.07E-01	8.36E-04	4.98E-05	1.07E+01	1.39E-01	1.06E+01	8.07E-02	6.45E+00
Benzo(a)anthracene	N/AV	N/AV	N/AV	N/AV	N/AV	N/AV	N/AV	N/AV	N/AV	N/AV	7.92E-03
Benzo(a)pyrene	N/AV	N/AV	N/AV	N/AV	N/AV	N/AV	N/AV	N/AV	N/AV	N/AV	7.79E-03
Benzo(b)fluoranthene	1.88E-03	6.32E-04	1.94E-03	6.27E-04	N/AV	N/AV	2.64E-01	8.34E-03	2.64E-01	8.48E-03	3.38E-02
Benzo(g,h,i)perylene	N/AV	N/AV	N/AV	N/AV	N/AV	N/AV	N/AV	N/AV	N/AV	N/AV	6.11E-03
Benzo(k)fluoranthene	7.91E-04	2.66E-04	8.24E-04	2.65E-04	N/AV	N/AV	2.45E-01	4.29E-03	2.44E-01	4.36E-03	1.18E-02
Bis(2-ethylhexyl)phthalate	8.01E-02	1.82E-03	6.24E-01	5.32E-01	N/AV	N/AV	N/AV	N/AV	N/AV	N/AV	N/AV
Cadmium	7.14E-02	2.81E-03	7.07E-02	2.53E-03	5.64E-05	1.53E-06	7.50E-01	1.13E-02	7.45E-01	1.06E-02	3.92E-01
Chrysene	3.31E-04	8.58E-05	3.53E-04	9.12E-05	N/AV	N/AV	3.14E-01	5.83E-03	3.14E-01	5.96E-03	8.67E-03
Copper	1.38E+00	1.25E-01	1.31E+00	1.16E-01	6.61E-04	4.36E-05	5.44E+01	4.92E-01	5.39E+01	3.40E-01	2.24E+01
Dibenzo(a,h)anthracene	N/AV	N/AV	N/AV	N/AV	N/AV	N/AV	N/AV	N/AV	N/AV	N/AV	7.67E-03
Fluoranthene	2.72E-03	4.86E-04	2.75E-03	4.91E-04	N/AV	N/AV	4.44E-01	1.80E-02	4.43E-01	1.82E-02	1.67E-02
Indeno(1,2,3-cd)pyrene	N/AV	N/AV	N/AV	N/AV	N/AV	N/AV	N/AV	N/AV	N/AV	N/AV	7.89E-03
Lead	5.30E-01	8.87E-02	4.75E-01	7.67E-02	4.87E-05	9.39E-06	4.32E+01	4.96E-01	4.28E+01	4.12E-01	6.31E+00
Mercury	1.93E-03	8.47E-04	1.97E-03	8.44E-04	5.82E-07	3.01E-08	2.05E-01	2.16E-03	2.04E-01	2.29E-03	N/AV
Nickel	8.99E-01	1.25E-01	8.82E-01	1.31E-01	4.46E-04	9.05E-06	2.27E+01	2.76E-01	2.24E+01	1.86E-01	5.22E+01
Phenanthrene	2.14E-03	1.60E-04	2.16E-03	1.71E-04	N/AV	N/AV	2.92E-01	1.89E-02	2.92E-01	1.91E-02	2.10E-02
Pyrene	4.26E-04	8.44E-05	4.62E-04	1.02E-04	N/AV	N/AV	5.10E-01	8.93E-03	5.09E-01	9.23E-03	1.44E-02
Total PCBs	1.30E-02	1.19E-02	1.30E-02	1.19E-02	N/AV	N/AV	6.53E-01	6.22E-03	6.52E-01	6.38E-03	N/AV
Tributyltin	6.85E-04	3.69E-04	7.06E-04	3.69E-04	N/AV	N/AV	2.02E-01	1.64E-03	2.01E-01	1.71E-03	N/AV
Zinc	2.44E+00	2.82E-01	2.29E+00	2.71E-01	1.20E-03	1.60E-04	1.04E+02	1.30E+00	1.02E+02	9.71E-01	4.74E+01

N/AV= Not Available

Table 3-7. Water and Sediment EEC Summary Table for the Heron Fledging Patch

Table 3-7. Water and Sediment EEC Summary Table for the Heron Fledging Patch

Chemical	Water (µg/L)						Sediment (mg/kg)				
	With CSOs		Without CSOs		Reference Site		With CSOs		Without CSOs		Refe
	Mean	Standard Error	Mean	Standard Error	Mean	Standard Error	Mean	Standard Error	Mean	Standard Error	Mean
1,4-Dichlorobenzene	4.16E-03	1.85E-03	4.05E-03	1.86E-03	N/AV	N/AV	2.89E-01	9.39E-03	2.90E-01	9.30E-03	N/AV
4-Methylphenol	2.82E-02	3.12E-03	2.64E-02	3.21E-03	N/AV	N/AV	2.48E-01	2.31E-02	2.51E-01	2.31E-02	N/AV
Arsenic	1.25E+00	4.68E-02	1.27E+00	6.28E-02	8.36E-04	4.98E-05	1.58E+01	1.09E-01	1.58E+01	1.39E-01	6.45E+00
Benzo(a)anthracene	N/AV	N/AV	N/AV	N/AV	N/AV	N/AV	N/AV	N/AV	N/AV	N/AV	7.92E-03
Benzo(a)pyrene	N/AV	N/AV	N/AV	N/AV	N/AV	N/AV	N/AV	N/AV	N/AV	N/AV	7.79E-03
Benzo(b)fluoranthene	1.60E-03	6.32E-04	1.81E-03	6.51E-04	N/AV	N/AV	3.19E-01	1.12E-02	3.24E-01	9.78E-03	3.38E-02
Benzo(g,h,i)perylene	N/AV	N/AV	N/AV	N/AV	N/AV	N/AV	N/AV	N/AV	N/AV	N/AV	6.11E-03
Benzo(k)fluoranthene	8.21E-04	3.11E-04	9.19E-04	3.18E-04	N/AV	N/AV	2.67E-01	7.59E-03	2.70E-01	6.78E-03	1.18E-02
Bis(2-ethylhexyl)phthalate	8.13E-02	1.80E-03	7.35E-01	6.24E-01	N/AV	N/AV	N/AV	N/AV	N/AV	N/AV	N/AV
Cadmium	7.79E-02	3.80E-03	7.76E-02	3.72E-03	5.64E-05	1.53E-06	9.39E-01	4.77E-02	9.21E-01	5.29E-02	3.92E-01
Chrysene	4.00E-04	9.51E-05	4.49E-04	1.12E-04	N/AV	N/AV	4.43E-01	1.46E-02	4.46E-01	1.39E-02	8.67E-03
Copper	1.19E+00	1.38E-01	1.13E+00	1.21E-01	6.61E-04	4.36E-05	6.67E+01	1.04E+00	6.60E+01	1.23E+00	2.24E+01
Dibenzo(a,h)anthracene	N/AV	N/AV	N/AV	N/AV	N/AV	N/AV	N/AV	N/AV	N/AV	N/AV	7.67E-03
Fluoranthene	3.78E-03	5.89E-04	3.93E-03	6.31E-04	N/AV	N/AV	5.85E-01	3.31E-02	5.91E-01	3.14E-02	1.67E-02
Indeno(1,2,3-cd)pyrene	N/AV	N/AV	N/AV	N/AV	N/AV	N/AV	N/AV	N/AV	N/AV	N/AV	7.89E-03
Lead	5.45E-01	1.04E-01	4.96E-01	9.11E-02	4.87E-05	9.39E-06	6.34E+01	1.49E+00	6.28E+01	1.66E+00	6.31E+00
Mercury	2.12E-03	1.03E-03	2.22E-03	1.03E-03	5.82E-07	3.01E-08	1.64E-01	3.06E-03	1.64E-01	2.93E-03	N/AV
Nickel	7.60E-01	1.33E-01	7.61E-01	1.40E-01	4.46E-04	9.05E-06	2.50E+01	2.65E-01	2.47E+01	3.34E-01	5.22E+01
Phenanthrene	3.70E-03	2.59E-04	3.71E-03	2.88E-04	N/AV	N/AV	3.35E-01	2.77E-02	3.36E-01	2.74E-02	2.10E-02
Pyrene	3.34E-04	4.18E-05	3.43E-04	4.66E-05	N/AV	N/AV	6.17E-01	2.00E-02	6.18E-01	1.96E-02	1.44E-02
Total PCBs	1.72E-02	1.62E-02	1.73E-02	1.62E-02	N/AV	N/AV	1.24E+00	1.07E-02	1.24E+00	1.03E-02	N/AV
Tributyltin	9.48E-04	4.97E-04	1.01E-03	4.97E-04	N/AV	N/AV	1.07E-01	4.18E-03	1.07E-01	4.05E-03	N/AV
Zinc	2.32E+00	2.83E-01	2.20E+00	2.87E-01	1.20E-03	1.60E-04	1.40E+02	1.95E+00	1.39E+02	2.32E+00	4.74E+01

N/AV= Not Available

Table 3-8. Water and Sediment EEC Summary Table for the Spotted Sandpiper Patch

Table 3-8. Water and Sediment EEC Summary Table for the Spotted Sandpiper Patch

Chemical	Water (µg/L)						Sediment (mg/kg)					
	With CSOs		Without CSOs		Reference Site		With CSOs		Without CSOs		Reference Site	
	Mean	Standard Error	Mean	Standard Error	Mean	Standard Error	Mean	Standard Error	Mean	Standard Error	Mean	Standard Error
1,4-Dichlorobenzene	5.31E-03	2.64E-03	5.18E-03	2.66E-03	N/AV	N/AV	6.91E-02	8.87E-04	6.91E-02	8.95E-04	N/AV	N/AV
4-Methylphenol	3.58E-02	5.90E-03	3.43E-02	6.00E-03	N/AV	N/AV	2.50E-02	8.82E-04	2.47E-02	9.69E-04	N/AV	N/AV
Arsenic	1.33E+00	1.35E-01	1.28E+00	1.04E-01	8.36E-04	4.98E-05	6.28E+00	5.88E-01	5.94E+00	4.29E-01	6.45E+00	1.04E+00
Benzo(a)anthracene	N/AV	N/AV	N/AV	N/AV	N/AV	N/AV	N/AV	N/AV	N/AV	N/AV	N/AV	9.48E-04
Benzo(a)pyrene	N/AV	N/AV	N/AV	N/AV	N/AV	N/AV	N/AV	N/AV	N/AV	N/AV	N/AV	6.51E-04
Benzo(b)fluoranthene	5.48E-03	2.03E-03	5.45E-03	2.01E-03	N/AV	N/AV	1.21E-01	1.30E-02	1.20E-01	1.31E-02	3.38E-02	0.00E+00
Benzo(g,h,i)perylene	N/AV	N/AV	N/AV	N/AV	N/AV	N/AV	N/AV	N/AV	N/AV	N/AV	N/AV	1.29E-03
Benzo(k)fluoranthene	2.09E-03	7.72E-04	2.09E-03	7.69E-04	N/AV	N/AV	9.99E-02	5.25E-03	9.94E-02	5.31E-03	1.18E-02	0.00E+00
Bis(2-ethylhexyl)phthalate	1.04E-01	6.52E-03	2.38E-01	1.14E-01	N/AV	N/AV	N/AV	N/AV	N/AV	N/AV	N/AV	N/AV
Cadmium	6.78E-02	8.95E-03	1.28E+00	1.04E-01	5.64E-05	1.53E-06	1.91E-01	1.67E-02	1.83E-01	1.31E-02	3.92E-01	1.54E-01
Chrysene	7.68E-04	2.12E-04	7.70E-04	2.09E-04	N/AV	N/AV	1.23E-01	4.49E-03	1.23E-01	4.73E-03	8.67E-03	1.22E-03
Copper	3.33E+00	3.86E-01	3.14E+00	3.16E-01	6.61E-04	4.36E-05	2.74E+01	1.62E+00	2.61E+01	1.17E+00	2.24E+01	3.89E+00
Dibenzo(a,h)anthracene	N/AV	N/AV	N/AV	N/AV	N/AV	N/AV	N/AV	N/AV	N/AV	N/AV	N/AV	1.24E-03
Fluoranthene	6.65E-03	1.45E-03	6.59E-03	1.43E-03	N/AV	N/AV	1.84E-01	1.58E-02	1.83E-01	1.62E-02	1.67E-02	4.58E-03
Indeno(1,2,3-cd)pyrene	N/AV	N/AV	N/AV	N/AV	N/AV	N/AV	N/AV	N/AV	N/AV	N/AV	N/AV	1.06E-03
Lead	1.41E+00	2.97E-01	1.25E+00	2.26E-01	4.87E-05	9.39E-06	1.97E+01	1.30E+00	1.89E+01	1.01E+00	6.31E+00	7.14E-01
Mercury	4.78E-03	2.47E-03	4.81E-03	2.47E-03	5.82E-07	3.01E-08	5.73E-02	5.53E-03	5.66E-02	5.66E-03	N/AV	N/AV
Nickel	1.62E+00	2.12E-01	1.53E+00	1.75E-01	4.46E-04	9.05E-06	1.16E+01	1.05E+00	1.09E+01	7.94E-01	5.22E+01	1.23E+01
Phenanthrene	5.08E-03	4.29E-04	5.14E-03	4.27E-04	N/AV	N/AV	9.51E-02	8.88E-03	9.43E-02	9.12E-03	2.10E-02	9.61E-03
Pyrene	1.85E-03	3.61E-04	1.93E-03	3.88E-04	N/AV	N/AV	1.85E-01	5.82E-03	1.83E-01	6.29E-03	1.44E-02	3.21E-03
Total PCBs	2.30E-02	1.97E-02	2.30E-02	1.97E-02	N/AV	N/AV	7.77E-01	1.71E-02	7.77E-01	1.70E-02	N/AV	N/AV
Tributyltin	1.75E-03	1.00E-03	1.75E-03	1.00E-03	N/AV	N/AV	3.83E-02	2.54E-03	3.78E-02	2.64E-03	N/AV	N/AV
Zinc	6.14E+00	9.78E-01	5.68E+00	7.90E-01	1.20E-03	1.60E-04	5.49E+01	4.56E+00	5.20E+01	3.45E+00	4.74E+01	6.61E+00

Table 3-9. Water and Sediment EEC Summary Table for the Bald Eagle Patch

Table 3-9. Water and Sediment EEC Summary Table for the Bald Eagle Patch

Chemical	Water (µg/L)						Sediment (mg/kg)					
	With CSOs		Without CSOs		Reference Site		With CSOs		Without CSOs		Reference Site	
	Mean	Standard Error	Mean	Standard Error	Mean	Standard Error	Mean	Standard Error	Mean	Standard Error	Mean	Standard Error
1,4-Dichlorobenzene	1.06E-03	6.61E-04	1.03E-03	6.64E-04	N/AV	N/AV	1.21E-01	5.58E-04	1.21E-01	5.56E-04	N/AV	N/AV
4-Methylphenol	5.82E-03	1.11E-03	5.43E-03	1.13E-03	N/AV	N/AV	1.56E-01	9.92E-04	1.56E-01	1.00E-03	N/AV	N/AV
Arsenic	1.32E+00	1.40E-01	1.32E+00	1.48E-01	8.36E-04	4.98E-05	1.07E+01	2.21E-02	1.07E+01	1.59E-02	6.45E+00	1.04E+00
Benzo(a)anthracene	N/AV	N/AV	N/AV	N/AV	N/AV	N/AV	N/AV	N/AV	N/AV	N/AV	7.92E-03	9.48E-04
Benzo(a)pyrene	N/AV	N/AV	N/AV	N/AV	N/AV	N/AV	N/AV	N/AV	N/AV	N/AV	7.79E-03	6.51E-04
Benzo(b)fluoranthene	4.27E-04	1.50E-04	4.46E-04	1.49E-04	N/AV	N/AV	5.42E-01	1.04E-02	5.42E-01	1.04E-02	3.38E-02	0.00E+00
Benzo(g,h,i)perylene	N/AV	N/AV	N/AV	N/AV	N/AV	N/AV	N/AV	N/AV	N/AV	N/AV	6.11E-03	1.29E-03
Benzo(k)fluoranthene	1.80E-04	6.35E-05	1.89E-04	6.35E-05	N/AV	N/AV	3.77E-01	3.69E-03	3.77E-01	3.67E-03	1.18E-02	0.00E+00
Bis(2-ethylhexyl)phthalate	6.83E-02	3.77E-04	6.62E-01	7.11E-01	N/AV	N/AV	N/AV	N/AV	N/AV	N/AV	N/AV	N/AV
Cadmium	6.71E-02	8.05E-04	6.68E-02	7.30E-04	5.64E-05	1.53E-06	1.26E+00	3.10E-03	1.26E+00	2.93E-03	3.92E-01	1.54E-01
Chrysene	7.03E-05	1.72E-05	7.42E-05	1.82E-05	N/AV	N/AV	6.02E-01	6.04E-03	6.02E-01	6.05E-03	8.67E-03	1.22E-03
Copper	6.78E-01	3.72E-02	6.49E-01	3.07E-02	6.61E-04	4.36E-05	7.27E+01	1.49E-01	7.26E+01	1.14E-01	2.24E+01	3.89E+00
Dibenzo(a,h)anthracene	N/AV	N/AV	N/AV	N/AV	N/AV	N/AV	N/AV	N/AV	N/AV	N/AV	7.67E-03	1.24E-03
Fluoranthene	6.56E-04	1.03E-04	6.62E-04	1.05E-04	N/AV	N/AV	9.24E-01	2.66E-02	9.24E-01	2.67E-02	1.67E-02	4.58E-03
Indeno(1,2,3-cd)pyrene	N/AV	N/AV	N/AV	N/AV	N/AV	N/AV	N/AV	N/AV	N/AV	N/AV	7.89E-03	1.06E-03
Lead	1.74E-01	2.64E-02	1.52E-01	1.93E-02	4.87E-05	9.39E-06	5.35E+01	1.62E-01	5.35E+01	1.40E-01	6.31E+00	7.14E-01
Mercury	8.75E-04	2.21E-04	8.86E-04	2.21E-04	5.82E-07	3.01E-08	3.59E-01	5.97E-04	3.59E-01	6.21E-04	N/AV	N/AV
Nickel	6.45E-01	1.19E-01	6.54E-01	1.37E-01	4.46E-04	9.05E-06	2.48E+01	6.42E-02	2.47E+01	4.56E-02	5.22E+01	1.23E+01
Phenanthrene	5.46E-04	3.59E-05	5.43E-04	3.81E-05	N/AV	N/AV	5.16E-01	2.89E-02	5.16E-01	2.89E-02	2.10E-02	9.61E-03
Pyrene	8.09E-05	1.47E-05	8.64E-05	1.75E-05	N/AV	N/AV	9.92E-01	9.97E-03	9.91E-01	1.00E-02	1.44E-02	3.21E-03
Total PCBs	7.24E-03	6.93E-03	7.27E-03	6.94E-03	N/AV	N/AV	4.22E-01	2.15E-03	4.22E-01	2.17E-03	N/AV	N/AV
Tributyltin	1.80E-04	1.01E-04	1.87E-04	1.01E-04	N/AV	N/AV	5.02E-01	7.02E-04	5.02E-01	7.03E-04	N/AV	N/AV
Zinc	9.98E-01	7.76E-02	9.38E-01	6.64E-02	1.20E-03	1.60E-04	1.15E+02	3.28E-01	1.15E+02	2.59E-01	4.74E+01	6.61E+00

N/AV= Not Available

Table 3-10. Water and Sediment EEC Summary Table for the River Otter Patch

Table 3-10. Water and Sediment EEC Summary Table for the River Otter Patch

Chemical	Water (µg/L)						Sediment (mg/kg)				
	With CSOs		Without CSOs		Reference Site		With CSOs		Without CSOs		Refe
	Mean	Standard Error	Mean	Standard Error	Mean	Standard Error	Mean	Standard Error	Mean	Standard Error	Mean
1,4-Dichlorobenzene	2.86E-03	1.45E-03	2.79E-03	1.45E-03	N/AV	N/AV	1.20E+00	1.97E-03	1.20E+00	1.89E-03	N/AV
4-Methylphenol	2.14E-02	3.24E-03	2.04E-02	3.25E-03	N/AV	N/AV	1.53E+00	1.16E-02	1.53E+00	1.19E-02	N/AV
Arsenic	1.19E+00	8.15E-02	1.21E+00	9.60E-02	8.36E-04	4.98E-05	3.37E+01	1.57E-02	3.37E+01	1.24E-02	6.45E+00
Benzo(a)anthracene	N/AV	N/AV	N/AV	N/AV	N/AV	N/AV	N/AV	N/AV	N/AV	N/AV	7.92E-03
Benzo(a)pyrene	N/AV	N/AV	N/AV	N/AV	N/AV	N/AV	N/AV	N/AV	N/AV	N/AV	7.79E-03
Benzo(b)fluoranthene	1.13E-03	4.87E-04	1.19E-03	4.86E-04	N/AV	N/AV	2.75E+00	5.50E-02	2.75E+00	5.51E-02	3.38E-02
Benzo(g,h,i)perylene	N/AV	N/AV	N/AV	N/AV	N/AV	N/AV	N/AV	N/AV	N/AV	N/AV	6.11E-03
Benzo(k)fluoranthene	4.44E-04	1.96E-04	4.70E-04	1.97E-04	N/AV	N/AV	2.14E+00	2.39E-02	2.14E+00	2.38E-02	1.18E-02
Bis(2-ethylhexyl)phthalate	8.13E-02	2.08E-03	6.21E-01	5.33E-01	N/AV	N/AV	N/AV	N/AV	N/AV	N/AV	N/AV
Cadmium	6.51E-02	2.86E-03	6.47E-02	2.70E-03	5.64E-05	1.53E-06	3.66E+00	1.05E-02	3.68E+00	7.12E-03	3.92E-01
Chrysene	2.60E-04	4.12E-05	2.69E-04	4.74E-05	N/AV	N/AV	3.92E+00	4.23E-02	3.93E+00	4.21E-02	8.67E-03
Copper	1.07E+00	8.67E-02	1.02E+00	8.79E-02	6.61E-04	4.36E-05	3.83E+02	2.31E-01	3.84E+02	1.84E-01	2.24E+01
Dibenzo(a,h)anthracene	N/AV	N/AV	N/AV	N/AV	N/AV	N/AV	N/AV	N/AV	N/AV	N/AV	7.67E-03
Fluoranthene	2.81E-03	3.53E-04	2.86E-03	3.55E-04	N/AV	N/AV	9.16E+00	2.59E-01	9.16E+00	2.59E-01	1.67E-02
Indeno(1,2,3-cd)pyrene	N/AV	N/AV	N/AV	N/AV	N/AV	N/AV	N/AV	N/AV	N/AV	N/AV	7.89E-03
Lead	3.66E-01	6.73E-02	3.29E-01	6.22E-02	4.87E-05	9.39E-06	4.47E+02	1.46E+00	4.48E+02	1.22E+00	6.31E+00
Mercury	2.04E-03	8.36E-04	2.09E-03	8.34E-04	5.82E-07	3.01E-08	1.57E+00	8.51E-04	1.57E+00	8.54E-04	N/AV
Nickel	7.52E-01	1.03E-01	7.49E-01	1.14E-01	4.46E-04	9.05E-06	2.43E+01	4.26E-02	2.43E+01	3.29E-02	5.22E+01
Phenanthrene	4.49E-03	2.27E-04	4.54E-03	2.29E-04	N/AV	N/AV	2.91E+00	1.59E-01	2.91E+00	1.60E-01	2.10E-02
Pyrene	1.82E-03	1.05E-04	1.83E-03	1.11E-04	N/AV	N/AV	6.75E+00	8.18E-02	6.75E+00	8.14E-02	1.44E-02
Total PCBs	1.36E-02	1.27E-02	1.36E-02	1.27E-02	N/AV	N/AV	3.15E+01	2.56E-01	3.15E+01	2.57E-01	N/AV
Tributyltin	5.61E-04	3.37E-04	5.90E-04	3.36E-04	N/AV	N/AV	1.01E+01	1.52E-02	1.01E+01	1.50E-02	N/AV
Zinc	1.97E+00	2.13E-01	1.87E+00	2.24E-01	1.20E-03	1.60E-04	2.82E+02	2.39E-01	2.82E+02	1.93E-01	4.74E+01

N/AV= Not Available

Table 3-11. Tissue EEC Data Used for Heron Fledgling, Heron, Spotted Sandpiper, Bald Eagle, and River Otter Exposure

	Perch (a)				Crab (b)													
	Duw Only		Duw & Elliott Bay		Dungeness Crab		Crab Hepatopancreas		Mussel (b)		Amphipods [c]		Combined Salmon (d)		Coho Salmon (d)		Chinook Salmon (d)	
COPC	Mean	SE	Mean	SE	Mean	SE	Mean	SE	Mean	SE	Mean	SE	Mean	SE	Mean	SE	Mean	SE
Metals/Metalloids																		
Cadmium	0.016	0.002309	0.023	0.003337	0.1413	0.09318	0.891	0.779	0.4895625	0.02873962	0.093	0.052	N/AV	N/AV	N/AV	N/AV	N/AV	N/AV
Copper	1.443667	0.377563	1.092167	0.230761	13.85	1.701911	35.75	7.15	1.28384375	0.066508232	263	36	0.6595	0.033619	0.620667	0.045035	0.698333	0.049482
Lead	0.161667	0.009615	0.1383	0.013685	0.19145	0.051471	0.1575	0.0245	0.4521875	0.035374678	6.315	1.105	0.027222	0.000856	0.027778	0.001292	0.026667	0.001143
Mercury	0.0779	0.005214	0.053267	0.011268	0.09165	0.011468	0.06075	0.00645	0.012428125	0.000479504	0.01335	0.00365	0.071928	0.006074	0.04145	0.001878	0.102406	0.006255
Nickel	0.183333	0.00857	0.185333	0.011526	0.087833	0.014363	0.335	0.095	0.536080125	0.04421687	0.6795	0.0915	N/AV	N/AV	N/AV	N/AV	N/AV	N/AV
Zinc	18.1	0.43589	17.61667	0.30921	51.03	4.316201	27.8	8.7	43.0565625	1.73515427	33.294	13.783	N/AV	N/AV	N/AV	N/AV	N/AV	N/AV
Organometallics																		
Tributyltin	0.152667	0.018095	0.1395	0.014621	0.047343	0.015093	0.05995	0.00075	0.05751875	0.008205248	0.0268	0.0092	N/AV	N/AV	N/AV	N/AV	N/AV	N/AV
Polychlorinated Biphenyls																		
Aroclor 1016	0.004	0	0.004	0	0.002875	3.04E-07	0.02	0	0.0065	0	0.004	0	N/AV	N/AV	N/AV	N/AV	N/AV	N/AV
Aroclor 1221	0.004	0	0.004	0	0.002875	3.04E-07	0.02	0	0.0065	0	0.004	0	N/AV	N/AV	N/AV	N/AV	N/AV	N/AV
Aroclor 1232	0.004	0	0.004	0	0.002875	3.04E-07	0.02	0	0.0065	0	0.004	0	N/AV	N/AV	N/AV	N/AV	N/AV	N/AV
Aroclor 1242	0.004	0	0.004	0	0.002875	3.04E-07	0.02	0	0.0065	0	0.004	0	N/AV	N/AV	N/AV	N/AV	N/AV	N/AV
Aroclor 1248	0.004	0	0.004	0	0.003933	0.001037	0.071	0.051	0.0065	0	0.0246	0.0021	N/AP	N/AP	N/AP	N/AP	N/AP	N/AP
Aroclor 1254	0.292667	0.048739	0.20545	0.045957	0.08385	0.022517	1.019	0.061	0.027328125	0.00316961	0.206	0.091	N/AP	N/AP	N/AP	N/AP	N/AP	N/AP
Aroclor 1260	0.203	0.028361	0.140267	0.031241	0.043167	0.016228	0.7275	0.2825	0.0065	0	0.10425	0.01575	N/AP	N/AP	N/AP	N/AP	N/AP	N/AP
Total PCBs	N/AV	N/AV	N/AV	N/AV	N/AV	N/AV	N/AV	N/AV	N/AV	N/AV	N/AV	N/AV	0.052342	0.004039	0.038472	0.003357	0.066211	0.005767
Semivolatile Organics																		
1,4-Dichlorobenzene	0.012	0	0.012	7.76E-11	0.008667	0.000667	0.012	0	0.008	0	0.012	0	N/AV	N/AV	N/AV	N/AV	N/AV	N/AV
4-Methylphenol	0.02	1.55E-10	0.02	0	0.014583	0.001083	0.02	0	0.029295	0	0.02	0	N/AV	N/AV	N/AV	N/AV	N/AV	N/AV
Benzo(a)anthracene	0.012	0	0.012	7.76E-11	0.0143	0.005539	0.012	0	0.015725	0.001582171	0.012	0	N/AV	N/AV	N/AV	N/AV	N/AV	N/AV
Benzo(a)pyrene	0.02	1.55E-10	0.023667	0.003667	0.019333	0.004656	0.02	0	0.0135	0	0.02	0	N/AV	N/AV	N/AV	N/AV	N/AV	N/AV
Benzo(b)fluoranthene	0.032	0	0.032	0	0.02325	0.00175	0.032	0	0.022171875	0.000671875	0.032	0	N/AV	N/AV	N/AV	N/AV	N/AV	N/AV
Benzo(g,h,i)perylene	0.02	1.55E-10	0.02	0	0.014583	0.001083	0.02	0	0.0135	0	0.02	0	N/AV	N/AV	N/AV	N/AV	N/AV	N/AV
Benzo(k)fluoranthene	0.032	0	0.032	0	0.02325	0.00175	0.032	0	0.0215	0	0.032	0	N/AV	N/AV	N/AV	N/AV	N/AV	N/AV
Bis(2-Ethylhexyl)phthalate	0.012	0	0.012	7.76E-11	0.008667	0.000667	0.012	0	0.023724438	0.00936454	0.54366	0.2847	0.435556	0.195396	0.427778	0.26732	0.443333	0.29282
Chrysene	0.012	0	0.012	7.76E-11	0.014	0.005241	0.012	0	0.02543125	0.00296393	0.012	0	N/AV	N/AV	N/AV	N/AV	N/AV	N/AV
Dibenzo(a,h)anthracene	0.032	0	0.032	0	0.02325	0.00175	0.032	0	0.0215	0	0.032	0	N/AV	N/AV	N/AV	N/AV	N/AV	N/AV
Fluoranthene	0.012	0	0.030317	0.012116	0.03365	0.015734	0.012	0	0.045956875	0.00321988	0.08736	0.06552	N/AV	N/AV	N/AV	N/AV	N/AV	N/AV
Indeno(1,2,3-cd)pyrene	0.02	0	0.02	0	0.014583	0.001083	0.02	0	0.0135	0	0.02	0	N/AV	N/AV	N/AV	N/AV	N/AV	N/AV
Phenanthrene	0.012	0	0.0302	0.01154	0.057867	0.035861	0.012	0	0.014635938	0.00115436	0.012	0	N/AV	N/AV	N/AV	N/AV	N/AV	N/AV
Pyrene	0.012	0	0.032267	0.014741	0.028683	0.01277	0.012	0	0.030989	0.00293714	0.0845	0.0725	N/AV	N/AV	N/AV	N/AV	N/AV	N/AV

Note: This is a revised table (Simmonds 2002) that replaces tissue data for the baseline CSO scenario previously presented in Tables 3-11 through 3-15 of the WQA (King County 1999)

a: Duwamish data were used for heron fledgling exposure; combined Duwamish and Elliott Bay data were used for heron exposure the remainder of the year, and for eagle and otter exposure

b: Combined Duwamish and Elliott Bay data; used for eagle and otter exposure

c: Duwamish data; used for sandpiper exposure

d: Duwamish data; used for eagle exposure

Table 3-16. Tissue Samples Used to Estimate Wildlife Exposure Concentrations (EECs)

Tissue Type	Number of Organisms Per Composite	Location	Number of Composite Samples	Tissue Analyzed
Shiner Perch *(Cymatogaster aggregate)*	10	Duwamish River Elliott Bay Port Susan	3 3 3	Whole body
Intertidal Amphipods *(Traskorchestia traskiana)*	2,000 approx.	Duwamish River Nisqually Delta	2 2	Whole body
Dungeness Crab *(Cancer magister)*	3	Duwamish River Elliott Bay Port Susan	2 4 3	Edible muscle & hepato-pancreas
Mussel *(Mytilus trossulus)*	50	Duwamish River Elliott Bay Totten Inlet	23 3 13	Soft parts
chinook Salmon *(Oncorhynchus tshawytscha)*	N/AV	Duwamish River	N/AV	Muscle
coho Salmon *(Oncorhynchus kisutch)*	N/AV	Duwamish River	N/AV	Muscle

N/AV = Not available

Table 4-10. Spotted Sandpiper Hazard Quotients

Chemical	Mean	90% Prediction Range	
		5th Percentile	95th Percentile
Baseline			
Copper	21.6	16.4	27.5
Lead	111.6	46.0	278.7
Total PCBs	2.5	1.5	3.7
Zinc	1.4	0.5	2.4
Without CSOs			

Copper	20.5	15.4	26.1
Lead	106.5	43.2	274.4
Total PCBs	2.5	1.5	3.7
Zinc	1.3	0.5	2.2
Reference Site (Nisqually Delta) (Dietary Risks Only)			
Copper	16.0	13.9	18.4
Lead	10.2	4.0	28.0
Total PCBs	0.2	0.1	0.4
Zinc	2.1	1.2	3.1p

Table 4-13. Average and 90% Prediction Interval Hazard Quotients for the River Otter Under Baseline and the Without CSO Condition

Chemical	HQ	90% Prediction Interval[a]	
		5th Percentile	95th Percentile
Baseline			
Arsenic	0.6	0.3	1.1
Lead	1.6	0.7	3.8
Without CSOs			
Arsenic	0.6	0.1	1.1
Lead	1.5	0.1	3.5
Reference (Dietary Exposure Only)			
Arsenic	0.2	0.1	0.3
Lead	<0.1	<0.1	0.1

[a] The 90% prediction interval represents the range between the 5th percentile and the 95th percentile HQs.

Table 4-16. Average and 90% Prediction Interval Hazard Quotients for the Bald Eagle Under Baseline and the Without CSO Condition

Chemical	HQ	90% Prediction Range	
		5th Percentile	95th Percentile
Baseline			
Lead	0.9	0.4	2.0
Without CSOs			
Lead	0.9	0.4	2.1
Reference (Dietary Exposure Only)			
Lead	0.1	<0.1	0.2

Table 4-19. Average and 90% Prediction Interval Hazard Quotients for the Great Blue Heron Under Baseline and the Without CSO Condition

Chemical	HQ	90% Prediction Range	
		5th Percentile	95th Percentile
Baseline Conditions			
Lead	0.9	0.5	1.8
Complete Year, Without Conditions			
Lead	0.9	0.5	1.7
Reference			
Lead	0.1	<0.1	0.1

Table 6-1. Water Column Selection Hierarchy

Freshwater Criteria	Washington State Surface Water Quality Standards (Title 173-201A WAC), or USEPA Ambient Water Quality Criteria (AWQC) (USEPA 1994), or Parametrix Criteria for Manganese and Cobalt (Parametrix 1997b), or

	Lowest Literature Value Divided by 20
Saltwater Criteria	Washington State Surface Water Quality Standards (Title 173-201A WAC), or USEPA Ambient Water Quality Criteria (USEPA 1994), or Parametrix Criteria for Manganese and Cobalt (Parametrix 1997b), or Lowest Literature Value Divided by 20, or Freshwater Criterion when no saltwater criterion/literature values are available

Table 6-2. Sediment Criteria Selection Hierarchy

Freshwater Criteria	USEPA Sediment Quality Criteria[138],or Ecotox Threshold (USEPA 1996), or Long and Morgan (1990), or Ingersoll et al. (1996), or Ontario Freshwater Sediment Guidance (Persaud et al. 1993), or Application Equilibrium Partitioning (EqP) to Chronic Water Quality Criteria after applying the 20 safety factor
Saltwater Criteria	Washington State Sediment Management Standards (Title 173-204 WAC), or Long et al. (1995), or Ecotox Threshold (USEPA 1996), or Application Equilibrium Partitioning (EqP) to Chronic Water Quality Criteria after applying the 20 safety factor

138 USEPA Sediment Quality Criteria are found in USEPA 1993a, 1993b, 1993c, 1993d, and 1993e.

REFERENCES FOR CHARTS AND TABLES

ATSDR. 1990. Draft toxicological profile for 4-methylphenol. US Department of Health and Human Services, Public Health Service, Agency for Toxic Substances and Disease Registry, Atlanta, GA.

ATSDR. 1991a. Draft toxicological profile for 1,4-dichlorobenzene. US Department of Health and Human Services, Public Health Service, Agency for Toxic Substances and Disease Registry, Atlanta, GA.

ATSDR. 1991b. Draft toxicological profile for cadmium. US Department of Health and Human Services, Public Health Service, Agency for Toxic Substances and Disease Registry, Atlanta, GA.

ATSDR. 1991c. Draft toxicological profile for cresols. US Department of Health and Human Services, Public Health Service, Agency for Toxic Substances and Disease Registry, Atlanta, GA.

ATSDR. 1991d. Toxicological profile for arsenic. Centers for Disease Control, Atlanta, GA.

Aulerich, RJ, Ringer RK,. Bleavins MR et al. 1982. Effects of supplemental dietary copper on growth, reproductive performance and kit survival of standard dark mink and the acute toxicity of copper to mink. J. Animal Sci. 55:337-343.

Bushy Run Research Center (BRRC). 1989. Two-generation reproduction study of p-cresol (CAS No. 106-44-5) administered by gavage to Sprague Dawley (CD) rats. Project Report 52-512. Unpublished data submitted to the Chemical Manufacturers Association Cresols Panel. Washington, DC.

Cain BW, Pafford EA. 1981. Effects of dietary nickel on survival and growth of mallard ducklings. Arch Environ Contam Toxicol 10:737-745.

Carlson GP, Tardiff RG. 1976. Effect of chlorinated benzenes on the metabolism of foreign organic compounds. Toxicol Appl Pharmacol 36:383-394.

Carmignani M, Boscolo P, Preziosi P. 1989. Renal ultrastructureal alterations and cardiovascular functional changes in rats exposed to mercuric chloride. Arch Toxicol Suppl 13:353-356.

Cecil HC, Bitman J, Lillie RJ, Fries GF, Verrett J. 1974. Embryotoxic and teratogenic effects on unhatched fertile eggs from hens fed PCBs. Bull Environ Contamd Toxicol 11:489-495.

Dunning JB Jr. 1993. CRC handbook of avian body masses. CRC Press, Inc., Boca Raton, FL. 371 pp.

Edens FW, Benton E, Bursian SJ, Morgan GW. 1976. Effect of dietary lead on reproductive performance in Japanese quail, *Coturniz conturnnix japonica*. Toxicol Appl Pharmacol 38:307-314.

Eisler R. 1988. Lead hazards to fish, wildlife, and invertebrates: a synoptic review. US Fish and Wildlife Service Biological Report 85(1.14). Patuxent Wildlife Research Center, US Fish and Wildlife Service, Laurel, MD.

Eisler R. 1986. Polychlorinated biphenyl hazards to fish, wildlife, and invertebrates: a synoptic review. US Fish and Wildlife Service Biological Report 85(1.7). Patuxent Wildlife Research Center, US Fish and Wildlife Service, Laurel, MD.

EPA. 1993a. Sediment quality criteria for the protection of benthic organisms: Acenaphthene. United States Environmental Protection Agency Office of Water and Office of Research and Development. Office of Science and Technology Health and Ecological Criteria Div. Washington, D.C. 20460. EPA-822-R-93-013.

EPA. 1993b. Sediment quality criteria for the protection of benthic organisms: Dieldrin. United States Environmental Protection Agency Office of Water and Office of Research and Development. Office of Science and Technology Health and Ecological Criteria Div. Washington, D.C. 20460. EPA-822-R-93-015.

EPA. 1993c. Sediment quality criteria for the protection of benthic organisms: Endrin. United States Environmental Protection Agency Office of Water and Office of Research and Development. Office of Science and Technology Health and Ecological Criteria Div. Washington, D.C. 20460. EPA-822-R-93-016.

EPA. 1993d. Proposed sediment quality criteria for the protection of benthic organisms: Fluoranthene. United States Environmental Protection Agency Office of Water and Office of Research and Development. Office of Science and Technology Health and Ecological Criteria Div. Washington, D.C. 20460. EPA-822-R-93-012.

EPA. 1993e. Sediment quality criteria for the protection of benthic organisms: Phenanthrene. United States Environmental Protection Agency Office of Water and Office of Research and Development. Office of Science and Technology Health and Ecological Criteria Div. Washington, D.C. 20460. EPA-822-R-93-014.

EPA. 1993f. Wildlife exposure factors handbook. EPA/600/R-93/187a. Office of Research and Development, US Environmental Protection Agency, Washington DC.

EPA. 1994. United States Environmental Protection Agency, Environmental Effects Branch, Health and Environmental Review Division, Office of Pollution Prevention and Toxics. A computer program for estimating the ecotoxicity of industrial chemicals based on structure activity relationships. User's Guide. EPA-748-R-93-002. U.S. EPA. 1996. Ecotox Thresholds. United States Environmental Protection Agency, Office of Solid Waste and Emergency Response, Publication 9345.0-12FSI, EPA 540/F-95/038.

EPA. 1996. Ecotox Thresholds. United States Environmental Protection Agency, Office of Solid Waste and Emergency Response, Publication 9345.0-12FSI, EPA 540/F-95/038.

Gaines TB, Linder RE. 1986. Acute toxicity of pesticides in adult and weaning rats. Fundam Appl Toxicol 7:299-308.

HEAST. Health effects assessment summary tables. 1995 Update. EPA/R-95-036. Office of Research and Development, Office of Emergency and Remedial Response, US Environmental Protection Agency, Washington, DC.

Heinz GH, Swineford DM, Katsma DE. 1984. High PCB residues in birds from the Sheboygan River, Wisconsin. Environ Monit Assess 4:155-161.

Hornshaw TC, Aulerich RJ, Johnson HE. 1983. Feeding Great Lakes fish to mink: effects on mink and accumulation and elimination of PCBs by mink. J Toxicol Environ Health 11:933-946.

Ingersoll, C.G., P.S. Haverland, E.L. Brunson, T.J. Canfield, E.J. Dwyer, C.E. Henke, N.E. Kemble, and D.R. Mount. 1996. Calculation and evaluation of sediment effects concentrations for the amphipod, *Hyallella azteca*, and the midge, *Chironomus riparius*. J. Great Lakes Res. 22: 602-623.

IRIS. 1998. Integrated Risk Information System: on-line computer database. National Center for Environmental Assessment, US Environmental Protection Agency. http://www.epa.gov/ncea/iris.htm.

Keplinger ML, Fancher OE, Calandra JC. 1971. Toxicologic studies with polychlorinated biphenyls. Toxicol Appl Pharmacol 19:402-403.

Lamb, JC 4th, Chapin RE, Teague J, Lawton AD, Reel JR. 1987. Reproductive effects of four phthalic acid esters in the mouse. Toxicol Appl Pharmacol 88:255-269.

Leach RM Jr, Wang K W-L, Baker DE. 1979. Cadmium and the food chain: the effect of dietary cadmium on tissue composition in chicks and laying hens. J Nutrition 109:437-443.

Lillie RJ, Cecil HC, Bitman J, Fries GF, Verrett J. 1975. Toxicity of certain polychlorinated and polybrominated biphenyls on reproductive efficiency of caged chickens. Poult Sci 54:1550-1555.

Long, E.R. and L.G. Morgan. 1990. The potential for biological effects of sediment-sorbed contaminants testes in the National Status and Trends Program. NOAA Technical Memorandum NOS OMA 52. National Oceanic and Atmospheric Administration, National Ocean Service, Office of Oceanography and Marine Assessment, Seattle, WA.

Long ER, McDonald DD, Smith SL, Calder FD. 1995. Incidence of adverse biological effects within ranges of chemical concentrations in marine and estuarine sediments. Env. Manag. 19:81-97.

MacKenzie KM, Angevine DM. 1981. Infertility in mice exposed *in utero* to benzo(a)pyrene. Biology of Reproduction 24:183-191.

Maxson SJ, Oring LW. 1980. Breeding season time and energy budgets of the polyandrous spotted sandpiper. Behaviour 74:200-263.

Melquist WE, Hornnocker MG. 1983. Ecology of river otters in west central Idaho. In: Kirkpatrick RL, ed, Wildlife monographs. Vol 83. The Wildlife Society, Bethesda, MD. 60 pp.

NAS. 1979. Symposium on pathobiology of environmental pollutants: animal models and wildlife as monitors. National Academy of Sciences, Washington, DC.

National Toxicology Program (NTP). 1987. NTP report on the toxicology and carcinogenesis studies of 1,4-dichlorobenzene in F344/N rats and B6C3F National Toxicology Program.

NRC. 1980. Mineral tolerance of domestic animals. Subcommittee on Mineral Toxicity in Animals, National Research Council, National Academy of Sciences, Washington, DC.

Oak Ridge National Research Laboratory (ORNRL). 1996. Toxicological benchmarks for wildlife: 1996 revision. ES/ER/TM-86/R3. Health Sciences Research Division, Oak Ridge, Oak Ridge National Research Laboratory, TN.

Pautzke CF, Meigs RC. 1940. Studies on the life history of the Puget Sound steelhead. Washington Department of Game, Olympia, WA.

Parametrix, Inc. 1997b. Pinal Creek WQARF site: Preliminary remediation performance goals for aluminum, cobalt, and manganese. Parametrix, Kirkland, Washington. 18 pp.+ appendices.

Peakall DB. 1974. Effects of di-N-butylphthalate and di-2-ethylhexylphthalate on the eggs of ring doves. Bull Environ Contam Toxicol 12:698-702.

Persaud, D., R. Jaagumagi, and A. Hayton. 1993. Guidelines for the protection and management of aquatic sediment quality in Ontario. Water Resources Branch, Ontario Ministry of the Environment and Energy. Queen's Printer for Ontario. ISBN 0-7729- 9248-7.

Ringer, Z. 1983. Toxicology of PCBs in minks and ferrets. In: D'Itri FM, Kamrin MA, eds, PCBs: Human and environmental hazards. Butterworth Publishing, Woburn, MA, pp 227-240.

Roberson RH, Schaible PJ. 1960. The tolerance of growing chicks for high levels of different forms of zinc. Poult Sci 39:893-896.

RTECS. 1995. Registry of Toxic Effects of Chemical Substances, on-line computer database. National Institute for Occupational Safety and Health, Centers for Disease Control and Prevention. http://www.cdc.gov/niosh/rtecs.html.

Schaefer EW Jr, Bowels WA Jr, Hurblet J. 1983. The acute and oral toxicity repellency and hazard potential of 998 chemicals to one or more species of wild and domestic birds. Arch Environ Contam Toxicol 12:355-382.

Scheuhammer AM. 1987. The chronic toxicity of aluminum, cadmium, mercury, and lead in birds: A review. Environ Pollut 46:263-295.

Schlatterer B, Coenen TMM, Ebert E, Grau R, Hilbig V, Munk R. 1993. Effects of Bis(tri-*n*-butyltin)oxide in Japanese quail exposed during egg laying period: an interlaboratory comparison study. Arch Environ Contam Toxicol 24:440-448.

Schlicker SA, Cox DH. 1968. Maternal dietary zinc, and development and zinc, iron, and copper content of the rat fetus. J Nutrition 95:287-294.

Schroeder HA, Mitchener M. 1971. Toxic effects of trace elements on the reproduction of mice and rats. Arch Environ Health 23:102-106.

Scott ML. 1977. Effects of PCBs, DDT, and mercury compounds in chickens and Japanese quail. Federation Proceedings 36:1888-1893.

Stahl JL, Greger JL, Cook ME. 1990. Breeding-hen and progeny performance when hens are fed excessive dietary zinc. Poult Sci 69:259-263.

Stalmaster, M. 1987. The bald eagle. Universe Books. New York, NY. 227 pp.

Stoewsand GS, Anderson JA, Gutenmann WH, Bache CA, Lisk DJ. 1971. Eggshell thinning in Japanese quail fed mercuric chloride. Science 173:1030-1031.

USFWS. 1964. Pesticide-wildlife studies, 1963: A review of fish and wildlife service investigations during the calendar year. FWS Circular 199. US Fish and Wildlife Service, Washington, DC.

White DH, Finley MT. 1978. Uptake and retention of dietary cadmium in mallard ducks. Environ Res 17:53-59.

Wren CD, Hunter DB, Leatherland JF, Stokes PM. 1987. The effects of polychlorinated biphenyls and methylmercury, singly and in combination on mink. II: Reproduction and kit development. Arch Environ Contam Toxicol 16:449-454.